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SUPRACONDYLAR FRACTURE OF THE HUMERUS *

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I make no apology for attempting to survey the treatment of so common a fracture as the supracondylar fracture of the humerus. The results of poor or indifferent treatment are so disastrous that a proper insight into the management of this fracture is essential not only for the surgeon, but the general practitioner who will very frequently be called upon to deal with it.

Supracondylar fracture of the humerus is an injury of childhood, caused by a fall on the outstretched hand and occasionally by a fall on the point of the elbow. The largest number of cases occur during the 6th year and 50% of cases occur between the ages of 5 and 7 years.

Clinical examination of a supracondylar fracture with the common displacement reveals the following features:

(a) The elbow is markedly swollen, the degree of swelling varying with the degree of displacement and the time which has elapsed since injury.

(b) The lower end of the anteriorly displaced upper fragment can often be palpated beneath the skin. The elbow is carried backwards and laterally owing to the backward and lateral displacement of the lower fragment.

(c) When there is over-riding of the fragments, measurements taken from the tip of the acromion process to the lateral epicondyle reveal appreciable shortening compared with the normal side. The relationship of the 3 bony prominences of the elbow, however, remains normal. This finding rules out the possibility of a posterior dislocation of the radius and ulna.

In all cases a very careful examination must be carried out immediately for nerve damage or vascular injury. The presence or absence of neurovascular injury must be recorded before any attempt at manipulation of the fracture is carried out. This will obviate any later question of such a complication having resulted from the manipulation.

X-ray examination will reveal not only the type of displacement and its extent but also whether there is an extension of the fracture line through the epiphyseal cartilage-plate of either of the condyles. The amount of serration of the fracture surface should also be noted because of its influence upon attempts at reduction and the maintenance of reduction of the displaced fragments. It is important to note the amount of medial or lateral displacement of the lower fragment. A lateral view will reveal the amount of anterior displacement of the upper fragment. The relationship of the distal fragment and the forearm, which are one, to the upper fragment in regard to the amount of rotation of the lower fragment is often demonstrable in this lateral view. The significance of the rotation of the lower fragment on the upper is usually better appreciated after the posterior displacement has been reduced, when the upper fragment can still be seen to project obliquely forward.

Approximately 20% of supracondylar fractures will not reveal any displacement; 75% will reveal posterior displacement of the lower fragment. Of this 75% group, in half the cases there will be no appreciable lateral or medial displacement; of the remaining half, marked lateral or medial displacement occurs, in equal proportions. The lower fragment is displaced anteriorly in 5% of cases; this fracture is usually caused by direct violence, due to a fall on the point of the elbow—the so-called flexion type of supracondylar fracture.

Complications. The danger of the supracondylar fracture lies in the associated damage to the brachial artery, with resultant spasm of the arterial tree to a greater or lesser extent, and the possible sequela of Volkmann's ischaemia.

The other danger of this fracture if the first complication has been obviated by adequate treatment, lies in the frequency with which limitation of movement of the elbow joint and deformity of the elbow joint results, in consequence of inadequate reduction of the displacement.

Other subsidiary complications may arise, e.g. ulnar, radial or median nerve palsy. These are usually caused by trauma from the fractured bone ends, sustained at the

* A paper presented at the South African Medical Congress, Pretoria, October 1955.

moment of impact. And finally, myositis ossificans may be a complicating factor as a result of injudicious passive movements.

TREATMENT

I wish to enlarge on the methods adopted (1) to avoid impending circulatory impairment, and (2) to procure satisfactory reduction of the fractured fragments, and a functionally useful elbow joint.

I have records of 4 cases of supracondylar fracture which presented with extreme displacement of bone ends, absence of the radial pulse, marked swelling of the elbow, coldness of the fingers and hand, and cyanosis of the fingers and hand in 2 cases and pallor of the fingers and hand in 2 cases.

This is the type of case where treatment is most urgent if a catastrophe is to be avoided. In 2 of the patients, a meal had been consumed 1½ hours before the accident. It is well known that the emptying time of the stomach after a fracture may be delayed up to 12 hours or even 24 hours. An attempt should immediately be made to pass a stomach tube. This will be found to be a most difficult procedure in a young child, but will usually be successful in inducing vomiting.

Where it is possible to pass a stomach tube, the stomach should be washed out, but here a note of warning must be added. Even if the stomach has been washed out, the greatest care must be exercised in the administration of the anaesthetic; I have on two occasions experienced further vomiting, with the danger of inhaling stomach contents into the trachea. Intratracheal intubation, with a cuff sealing off the lumen of the trachea, is by far the safest way to administer the anaesthetic.

The displacement of the fracture ends is reduced and the circulaion of the limb assessed.

In this type of case the displacement is always extreme, and it appears to me that the more extreme the displacement, the easier is the reduction and the more stable are the fragments after reduction. One can almost compare this type of supracondylar displacement with a dislocation which stays 'put' once it is reduced.

In all 4 cases reduction of the displacement caused immediate improvement in the colour of the hand and fingers, and the return of fairly brisk blanching to pressure of the finger-tips. In 2 cases the radial pulse remained completely impalpable, returning in one case after 7 days, and in case 2, a weak radial pulse could only be felt 6 weeks later. In none of these cases was there any evidence later of a Volkmann's ischaemia or motor or sensory impairment. The return of the radial pulse *per se* would appear to be of little significance compared with the return to the fingers and hand of normal colour and blanching.

It is conceivable that a case may arise where severe damage done to the brachial artery causes extreme spasm of the collateral arterial tree. In such a case it is possible that reduction of the displacement will not cause an adequate circulatory return to the fingers, and sympathetic anaesthetization followed by exploration and stripping of the artery or arteriectomy may be necessary. It would, however, be reasonable to say that, in the vast majority of cases, immediate and adequate reduction of the displacement by non-operative measures will be successful in restoring the circulation to the limb. It is important in this respect not to flex the elbow too much, as with increasing swelling of the elbow joint within the next 48 hours, impairment of the circulation may again arise, should the elbow be too much flexed, or should the brachial artery or collateral circulation be impeded by plaster of paris.

I make a particular point of never applying a circular plaster to the arm. A posterior slab alone is applied in the maximum degree of flexion consistent with the efficiency of the circulation. I also make a particular point of having no bandaging in the cubital fossa, but applying the retaining gauze bandage from forearm to arm across the cubital fossa, thus leaving the space free of any bandage whatever in front of the cubital fossa.

The arm is then elevated and the circulation watched carefully. At the slightest sign of circulatory impairment or excessive swelling any constricting bandage is divided and the flexion of the elbow diminished. The circulation of the limb takes pride of place and all other considerations, such as position of the fracture, are of secondary importance.

Reduction of Fragments. In my experience open reduction has never yet been necessary. Reduction must be by manipulative methods; it is an almost universal experience that open reduction leads to a good radiological but poor functional result.

Reduction should be carried out urgently before swelling of the elbow occurs because, once there is much swelling of the elbow, reduction is difficult and the retention of the reduced position still more difficult since the surgeon is unable to flex the elbow to a sufficient extent.

Anatomical reduction is most desirable, for it will lead to bony union, with the return of normal function, in the shortest possible time; but, though most desirable, it is very frequently not possible. The following conditions, however, must be fulfilled:

1. There must be no angulation in the antero-posterior or lateral views. Angulation in the A.P. view will lead to either cubitus varus or cubitus valgus deformity of the elbow joint with its resultant sequelae of delayed ulnar neuritis and cosmetic unsightliness. Angulation in the lateral view will lead to limitation of flexion or extension of the elbow joint.

2. There must be no residual rotation of the lower fragment. If angulation and rotation at the site of fracture are avoided, the functional result will be good.

In children under the age of 10 years, where most of these fractures occur, extensive re-modelling takes place at the site of union with the passage of years, resulting in marked anatomical improvement. The anterior spur becomes smaller and relatively higher up as growth proceeds at the lower humeral epiphysis, in this way diminishing the bony block to flexion; likewise the medial and lateral spurs become smaller or completely absorbed. Angulation at the site of fracture, however, is never corrected by re-modelling, nor is unreduced rotation.

Many supracondylar fractures after initial good reduction will show change of position after subsidence of swelling and will require to be re-manipulated. This should not be delayed longer than 5 to 7 days because

after this time the fracture rapidly shows signs of commencing union, and sub-periosteal new-bone formation may be seen radiologically as early as 8—10 days.

A significant relationship exists between the character of the line of fracture and the reduction. A serrated transverse fracture properly reduced will remain in alignment if immobilized with the forearm flexed at a right angle, irrespective of the amount of supination or pronation. On the other hand, an oblique fracture line with an anteriorly displaced upper fragment presents a serious problem, for the displacement frequently recurs after the reduction because of the lack of serrations on the fractured surface.

Immobilization of the elbow in flexion with semi-pronation of the forearm relaxes the pronator-flexor group of muscles and lessens the tendency to re-displacement. The elbow, however, must not be flexed until traction has been exerted on the fracture fragments and over-riding has been overcome. Any medial or lateral displacement which may be present is then corrected. Persistent rotational displacement can be overcome by fixing the upper fragment manually or by abduction of the shoulder during the manipulative procedure. The rare type of 'flexion' supracondylar fracture is immobilized with the elbow in extension.

Satisfactory reduction of the fracture having been obtained, the elbow is immobilized in the manner already described. Immobilization is continued for 4—6 weeks until union is complete. Movements of the elbow are then allowed to recover slowly of their own accord, with particular avoidance of passive movements, passive stretching and massage of the elbow. The carrying of heavy weights and hanging from overhead beams are two forms of passive stretching which must be rigorously avoided.

It has been my experience that by following these simple precepts a satisfactory result can be procured.

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ABSTRACT : UITTREKSEL

The Coronary Artery Problem. Claude S. Beck (1955): Geriatrics, **10,** 501.

The problem of coronary artery disease becomes the two-part problem of (1) the electrically unstable heart and (2) insufficient blood-supply to the myocardium. Uneven distribution of oxygen, in either the well or poorly oxygenated heart, causes differences in electrical potential sufficient at times to destroy the coordinated mechanism and produce fibrillation.

Loss of blood supply in the progressively occluded heart, 'causes irreversible damage from fibrosis,' with ultimate failure and death. No means are at present known to restore damaged muscle; heart research at this time ought to be concentrated on the problem of electrical stability.

Yater[1] has shown that one-third of all deaths from coronary artery disease occur in hearts that show no muscle damage. More than one half of the total deaths occur in hearts still anatomically capable of functioning, in which electrical instabillty is combined with heart-muscle damage. Only 10% of coronary-artery deaths occur in electrically stable hearts whose failure is due to heart-muscle damage alone. Uniformity of blood distribution is more important than quantity of blood.

Zoll *et al*[2] have shown that 9% of all human hearts possess natural intercoronary arteries (probably from birth), channels in which blood can 'move over' from one artery to another. More than one-half of the patients who survive 75% (or greater) occlusion develop intercoronaries, and all patients surviving 100% chronic closures have such channels. Dr. Beck proposes a surgical method to produce intercoronaries artificially by means of abrasion.

In experiments made on 67 dogs, by Beck's associate Leighninger, it was discovered that 5 c.c. per minute, or 300 c.c. per hour, of back flowing blood are necessary for an animal to survive ligation and resultant descending-artery occlusion; 6.5 c.c. of blood per minute are needed to survive circumflex-artery occlusion; all animals survived with large infarcts. Spontaneous recovery occured in those animals having 5 c.c. of back-flowing blood (10%) after descending-artery occlusion and in 58% of animals with 6.5 c.c. of back-flowing blood after circumflex-artery occlusion.

Performing the Beck 1 operation on a group of normal dogs, with surgical procedure consisting of abrasion, application of inflammatory agent such as asbestos, partial occlusion of the coronary sinus, and grafting parietal pericardium and mediastinal fat to the heart surface—prior to arterial ligation—produced a recovery rate of 73·4%. The size of infarct was reduced by 60-70%. 'The Beck 1 operation perfuses the circumflex arterial bed with 282 c.c. of blood per hour . . . available immediately after arterial occlusion . . . This amount of blood lowers mortality by reducing oxygen differential and electrical potentials, and reduces the infarct by preserving the viability of heart muscle.'

Selection of human patients for the Beck 1 operation is based on several criteria. In all cases, the operation is thought of as a prohylactic measure. It has been performed on a well patient with heavy familial history of coronary disease. Other suitable candidates include: patients with coronary insufficiency without infarct; patients with occlusion but without infarct; and patients with one or more infarcts, normal heart size, and ability to work. There is little or no operative risk with such patients. Patients with one or more infarcts, slight enlargement, and ability to work have provided most of the surgical cases to date. While candidates with more severe cardiac symptomatology are considered operable, they are regarded as 'salvage' cases and have a higher mortality risk.

In the last 100 operative cases, mortality was 6-8%, with 2 patients dying of thoracotomy alone. Nine out of 10 patients are 'improved to the point where they have little or no pain and where they can return to work.' It is emphasized that the operation does not prevent future occlusion, but 'the intercoronaries produced by operation lessen the effects of the occlusion when it occurs.'

The research reported is based on 5,000 operations on coronary vessels in dogs, and 260 heart operations on human patients.

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UNION DEPARTMENT OF HEALTH BULLETIN

Department of Health Bulletin. Report for the 7 days ended 16 Feburary, 1956.

Plague, Smallpox, Typhus Fever: Nil.

Epidemic Diseases in Other Countries.

Plague: Nil.

Cholera: Nil.

Smallpox in Kabul (Afghanistan); Moulmein, Rangoon (Burma); Phom-Penh (Cambodia); Ahmedabad, Bombay, Delhi, Madras, Visakhapatnam (India); Chittagong, Dacca (Pakistan); Hué (Viêt-Nam).

Typhus Fever: Nil.

South African Medical Journal

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VAN DIE REDAKSIE

DIE PYN VAN DIABETIESE SENUWEESIEKTE

'n Opvallende siekteteken by baie suikersiektepasiënte is pyn, wat dikwels aan senuweesiekte toegeskryf kan word alhoewel bewyse van neurologiese aantasting meer dikwels met die ondersoek gevind word (die enkel ruk nie, die voet is nie vir trilling gevoelig nie) as wat van die pasiënt se klagtes vermoed sou word. Die uitstaande simptoom is pyn in die bene. Die pyn is kenmerkend kwaai en aanhoudend, dikwels onafgebroke, alhoewel elke nou en dan deurskiet met krampe, swak gelokaliseer en weerskantig, erger saans en versag deur beweging, nie deur houding of hitte geaffekteer nie, en dit gaan gepaard met abnormale gevoeligheid van die ‚pins and needles' tipe. Die vel en die spiere mag uitermate gevoelig wees, selfs die gewig van lakens en komberse kan ondraaglik wees.

Pyn, ischemies vaskulêr in oorsprong, mag ook met rustye voorkom, maar op tipiese wyse word dit erger met verandering van die weer of liggaamshouding in hierdie gevalle is 'n verswakte bloedsomloop, met geen kloppings nie, byna altyd vanselfsprekend—dikwels met dreigende gangreen.

Dit mag oor senuweepyne wees wat die onbehandelde suikersiektelyer kla. In so 'n geval kan spoedige verligting (soos bv. in die geval van pruritis vulvae) nie belowe word nie, maar die waarskynlikheid van verligting kan verduidelik word en uiteindelike verlossing kan byna verseker word. Hierdie vroeë gevalle toon miskien geen abnormale fisiese tekens dat die senuwees betrokke is nie.

Dit gebeur af en toe dat hierdie tipiese pyn begin sodra die hiperglisemie (in 'n matige of ernstige geval van suikersiekte) deur die insulien beheer is. Een jong man moes onlangs 'n week of wat na sy eerste ontslag weer in die saal toegelaat word omdat die pyn so kwaai was dat hy nie kon slaap nie; hy was maer en bleek en het vinnig gewig verloor. Sy bloedsuiker was nie abnormaal laag nie, tog het sy pyn alleen bedaar wanneer die insulien verminder was en die urientoetse groen en geel getoon het. Hy is nou heeltemal sonder pyn en goed beheer.

Ander pasiënte ondervind pyn eers na jare. Roetinemaatreëls en bevredigende beheer van die suikersiekte

EDITORIAL

PAIN ASSOCIATED WITH DIABETIC NEUROPATHY

Pain is a prominent symptom in many cases of diabetes. It may often be ascribed to neuropathy, though evidence for neurological involvement is more commonly found on examination (absence of ankle jerks, loss of vibration sense in the feet, etc) than suspected by reason of the patient's complaints. The outstanding symptom is pain in the legs. This pain is characteristically of severe aching type, often continous though punctuated by shooting spasms, poorly localized and bilateral, worse at night and eased by action, unrelated to posture or warmth, and accompanied by paraesthesiae of 'pins and needles' type. Both skin and muscles may be extremely tender; even the pressure of bedclothes may be unbearable.

Pain of ischaemic vascular origin may also occur at rest, but is typically related to change of posture and of temperature; in these cases impaired circulation, with absence of pulsations, is almost always obvious, often with threatened gangrene.

Neural pain may be the presenting complaint in any untreated diabetic. In such a case one cannot promise rapid relief (as for instance one can from pruritus vulvae), but one can explain the likelihood of such relief and can certainly almost promise eventual remission. In these early cases there may be no abnormal physical signs of neurological involvement.

Occasionally this typical pain starts just as the hyperglycaemia (in a moderate or severe diabetic) is brought under control by insulin. One young man recently required readmission to the ward a week or so after his initial discharge, because of pain so severe that sleep was impossible; his appearance was haggard and his weight had fallen rapidly. His blood sugar was not abnormally low, yet his pain subsided only when insulin was reduced and the urine tests showed green and yellow. He is now quite free from pain and again well-controlled.

In other patients the pain occurs only after years. In one middle-aged man it was unrelieved by good control

het aan een middeljarige man geen verligting gebring nie, en was die pyn so kwaai dat chordotomie nodig was.

Kramppyne in die kuit (of minder algemeen in die dy of die boud) wat na inspanning voorkom en wat spoedig deur rus verlig word, is die kenmerkende vroeë simptoom van aarbeskadiging (die sogenoemde *‚intermittent claudication'*). Somtyds egter toon sorgvuldige toetse, self met gespesialiseerde metodes, geen beskadiging in die bloedsomloop nie maar definitiewe tekens van perifërale senuweeontsteking. Vermoedelik is bloedvatvernouingskreupelheid soms neuropaties van oorsprong.

Maagpyn wat by suikersiekte voorkom mag te wyte wees aan alvleesklierontsteking, ketose, 'n onlangs vergrootte lewer met verrekte kapsuul, of, baie selde, aan hipoglisemie. As hierdie kondisies uitgeskakel kan word bly daar nog 'n paar pasiënte oor van 'n ernstige, vroeë tipe wat insulien nodig het by wie maagpyn 'n voreë klagte of selfs 'n terugkerende episode is, dikwels met vomering gepaard. Hierdie aanvalle mag plaasvind wanneer die suikersiekte goed onder beheer is en wanneer daar geen tekens van hipoglisemie is nie. Dit kan vir beide geneesheer en pasiënt hoofbrekens besorg. Miskien is dit te wyte aan senuweesiekte van die abdominale outonomiese stelsel. Staan hul enigsins in verband met die ‚maagkrisisse' van uittering? Hul patroon is baie eenders. Die gereelde terugkeer van die pyn suggereer selfs ‚abdominale epilepsie' en behandeling met epanutin is al op die proef gestel.

Pyn in die vingertoppe gepaard met parestese en gewoonlik sonder fisiese tekens mag tergelyktydig met die hewiger pyne in die bene ondervind word. Andersins is dit twyfelagtig of pyn in die arms 'n teken van diabetiese senuweesiekte is. Weliswaar dat klagtes daaroor dikwels in die suikersiektekliniek aangehoor word; om dit as senuweeontsteking te bestempel is te gemaklik. Afgesien van kortstondige ‚fibrositis', of wat ook al, is die pyn meesal aan subakromiale slymbeursontsteking toe te skryf en X-straalbehandeling bring gewoonlik verligting.

Ander pyne ook, selfs in die bene, moet nie te geredelik aan senuweesiekte toegeskryf word nie. So is eensydige heupjig nie diabeties nie; inderdaad behoort baie sorgvuldige aandag geskenk te word aan enige mononeuritis of gelokaliseerde senuwee-aandoening van anatomiese beperking alvorens dít as ‚diabetiese neuritis' te bestempel.

Ortopediese voetmoeilikhede van verskeie tipes en selfs ingroeiende toonnaels word somtyds senuweesiektes genoem. Nagkrampe en ‚voete wat spring' kom dikwels by bejaardes voor wat nie aan suikersiekte ly nie, en behandeling met kina en soms klein dosisse barbituraat is meer geslaagd as met Vitamien-B.

Daar is geen bewys nie dat Vitamien-B al ooit 'n pasiënt met diabetiese senuweesiekte gehelp het nie. Die suikersiektelyer se dieet moet natuurlik nooit so abnormaal wees om in hierdie vitamien kort te skiet nie. Dit is twyfelagtig of enig iets behalwe tyd en goeie beheer van die suikersiekte die senuweesiekte raak. Vitamiene, lewer, B.A.L. en die res laat die meeste suikersiekte-navorsers onoortuig. In elk geval as aktiewe maatreëls nodig geag word, kan miskien aan die hand gedoen word dat die goedkoopste eerste probeer word.

of the diabetes and routine measures and was so severe that cordotomy was performed.

Cramping pain in the calf (or less commonly in the thigh, or buttock) on exertion, with rapid easement on rest, is the characteristic early symptom of arterial impairment (so-called 'intermittent claudication'). Sometimes, however, careful tests, even with specialized methods, indicate no circulatory abnormalities, but definite signs of peripheral neuritis. Presumably 'intermittent claudication' on occasion is neuropathic in origin.

Abdominal pain related to diabetes may be due to pancreatitis, ketosis, a recently enlarged liver with stretched capsule or, rarely, hypoglycaemia. When these conditions can be ruled out there remain some patients of the severe, 'young', insulin-requiring type in whom abdominal pain is an early complaint or even a recurrent episode, often accompanied by vomiting. These attacks may occur when the diabetes is under good control and when hypoglycaemia is not present. They can be very trying for both the patient and the doctor. Perhaps they are caused by neuropathy of the abdominal autonomic system. Could they be related to the 'gastric crises' of tabes? Their pattern is very similar. Their periodicity even suggests 'abdominal epilepsy' and treatment with Epanutin has been tried.

Finger-tip pain, with paraesthesiae and usually without physical signs may occur in conjunction with the more severe leg pain. Otherwise it may be doubted whether pain in the arms is a manifestation of diabetic neuropathy. Certainly the complaint is common in the diabetic clinic; it is too easy to call it 'neuritis'! Apart from transitory 'fibrositis' or what-you-will, the commonest cause for the complaint in general is probably a subacromial bursitis, usually amenable to X-ray therapy.

Other pains, too, even in the legs, must not readily be ascribed to neuropathy. Thus unilateral 'sciatica' is not diabetic; in fact any mononeuritis or localized neural involvement of anatomical delineation must be very carefully considered before being dubbed 'diabetic neuritis'.

Orthopaedic foot-strain of various types and even ingrowing toenail, are sometimes called neuropathy. Nocturnal cramps and 'jumping feet' of the elderly are common in non-diabetics and respond better to quinine and sometimes a little barbiturate than to vitamin B1!

There is no evidence that vitamin B1 ever helped a patient with diabetic neuropathy. A diabetic patient's diet should never be so abnormal as to be deficient in this vitamin. It is doubtful whether anything other than good diabetic control and time affects the neuropathy itself. Vitamins, liver, B.A.L. and the rest have left most workers in diabetes unconvinced. Anyway, if some active measure is considered essential it might be advised that the least expensive should be tried first.

PITYRIASIS ROSEA

A REVIEW OF ITS CLINICAL ASPECTS AND A DISCUSSION OF ITS RELATIONSHIP TO PITYRIASIS LICHENOIDES ET VARIOLIFORMIS ACUTA AND PARAPSORIASIS GUTTATA*

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Pityriasis rosea (P.R.) is a relatively common skin disease of world-wide distribution. The common macular type was first described by Gibert in 1860, and since this time many modifications and varieties have been added. Historical details can be found in an article by Percival[1] and in the *Nouvelle Pratique Dermatologique*.[2]

P.R. is a self-limiting disease of young people with certain characteristics which are clearly seen in many cases. It often begins with a solitary (large) lesion, the herald patch, which appears almost always on the body and is followed after a few days to a few weeks by a secondary disseminated eruption of smaller lesions (often in hundreds) over the body and limbs. The secondary rash appears in crops over a week or two after which the lesions gradually heal and disappear without scarring in 6-12 weeks. Symptoms are mild and usually unimportant; itch is often present in the early days but is rarely severe or persistent. The course of P.R. has been compared to that of early syphilis and the exanthemata. Most patients are children or young people. Second attacks are very rare. The disease is endemic and most cases are seen, the world over, during the colder months of the year. Apart from seasonal fluctuations there are also times when case incidence reaches proportions deserving the term epidemic. The number of occasions on which more than one case has occurred in a household or in other closely associated people is not large.

The course of a typical case of the macular variety is as follows. The herald patch appears on the body as a ringed or oval plaque, from 2-5 cm. in diameter, which may enlarge peripherally still further. The border is red, finely scaling and slightly elevated; the centre is flat and pink or yellowish-brown and healing begins there before it does in the edges. The herald patch, especially if it is on the back, may not be noticed by the patient.

A few days to a fortnight after the herald patch appears the secondary rash begins to erupt and lesions appear in crops over the body, neck, upper arms and thighs for about a fortnight. The secondary lesions are usually of two main types, (*a*) small pink finely-scaling macules, 2-10 mm., and (*b*) round or oval nummular lesions (medallions), 10-30 mm., with a pink, slightly elevated, finely-scaling edge and a flat or depressed, yellowish, atrophic-looking centre. The skin in the centre is like tissue paper and may also scale off. These lesions resemble the herald patch and may run together to form large circinate plaques. Some of the macular lesions may alter to the nummular type.

Healing begins after 2-4 weeks, first in the lesions that appeared earliest, and is usually complete by 6-12 weeks when all scales have fallen off leaving an unblemished skin. Rarely the sites of the lesions remain slightly depigmented or hyperpigmented for a short time after healing is complete.

The histological features, to be detailed later, are those of an eczematide.

Although most cases of P.R. (78% of 1,556 cases, according to Benedek[3]) conform more or less to the above description a great variety of untreated cases differing in behaviour and clinical features from the macular type have been described. A form of classification, modified from that of Klauder,[4] is shown in Table I.

The Herald Patch

The herald patch, usually solitary, is almost always found on the body; and there are no particular sites of election. It is rarely seen away from the covered areas of the body but cases are described where it has been found on the face, palms, soles or genitals (even the glans penis) and I have recently seen a case where it occurred on the scalp. It is usually 2-5 cm. in diameter (larger than the secondary lesions) but may be smaller or very much larger (giant herald patch). It may pass unnoticed by the patient and be identified only by its size, or it may apparently be absent. Rarely there may be 2 or even 3 herald patches. Atypical lesions, although less common than in the secondary eruption, are sometimes seen.

When the herald patch is seen before the secondary rash has appeared it is frequently diagnosed and treated as tinea corporis; and the stronger fungicides such as iodine and Whitfield's ointment may cause it to become eczematous. If the lesion is in the axilla or groin it may be very difficult to differentiate from tinea axillaris or cruris.

The herald patch remains solitary, in most cases, for 4-14 days, but the secondary rash may appear with it or only after a much longer delay of as much as 6-7 weeks. There are authentic cases where the herald patch has not been followed by a secondary rash.

The Secondary Rash

The lesions of the secondary rash are usually widely and symmetrically distributed over the body, neck and adjacent parts of the limbs; those on the back and in the axillae often lie along the lines of the ribs. The palms and soles are very rarely affected. Degos,[5] like many other observers, states that pityriasis rosea 'almost always spares the face and always spares the scalp'. This is not correct; Haxthausen[6] has drawn attention to the fact that the scalp is usually affected in children and the face

* A paper presented at the South African Medical Congress, Pretoria, October 1955.

and scalp are quite often affected in the Bantu, children and adults, of South Africa (Figs. 1 and 2). Scalp lesions are, of course, more easily visible in the Bantu because of the short hair. The secondary rash may remain relatively localized around the herald patch on any part of the body. I have several times seen cases in men in which the lesions were largely confined to the untanned bathing-trunk area, only faint fleeting lesions being

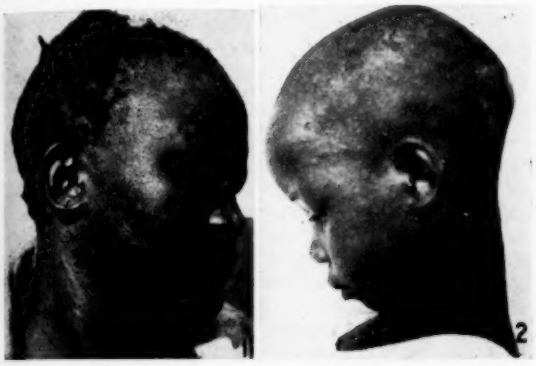

Fig. 1. Pityriasis rosea of the face in a Bantu.
Fig. 2. Pityriasis rosea of the scalp in a Bantu child.

present elsewhere; and this phenomenon has been noted by others. This beneficial effect of sunlight may well explain the relative rarity of lesions on the face and extremities.

Apart from the macular lesions those most commonly seen are vesicular, urticated and small papular. Vesicles, in untreated cases, are rarely larger than pinhead size, but occasionally the description varicelliform may be warranted. Pustules are also sometimes seen. When urtication occurs the oedema is usually confined to the edges of the lesions and very rarely is urticaria simulated. Among the papular forms the lichen planus-like variety is common (in South Africa at least), and tiny lichenoid papules are thickly set in the edges of the lesions and scattered in the immediate surroundings.

I have included in my classification the *Pityriasis circiné et marginé* of Vidal. Although Gougerot[2] and some other observers classify it separately the majority consider it simply a type of P.R. in which the lesions are fewer, larger and of longer duration. They may be distributed mainly in the axillary and crural regions, resembling closely the lesions of tinea cruris et axillaris.

The other varieties shown in the table need no further explanation. It is rarely that one sees a case in which all the lesions are of some unusual type; in most cases among the atypical lesions are some typical macular or nummular lesions, and perhaps the herald patch, to point to the diagnosis.

Healing, in an uncomplicated case, takes place without leaving any scar. Post-lesional depigmentation or hyperpigmentation of unimportant degree may remain. Hyperpigmentation is relatively common in the Bantu and may take some months to fade entirely.

Vesicular, bullous and erosive lesions of the buccal mucosa have been reported by a few observers and Wile[7] has described lesions on the vulva. I have seen only one case with mucosal (buccal) lesions; tiny haemorrhages and pinhead erosions were scattered over the cheeks in a European with severe diffuse vesicular P.R. affecting also the face.

The secondary rash appears in crops over a few weeks and all lesions have healed, in the average case, in from 6-12 weeks. There are, however, cases in which fresh crops of lesions continue to appear for very much longer. The case of longest duration that I have seen lasted just over a year; the clinical picture was of a typical macular P.R.

Differential Diagnosis

In a typical case of the macular variety with an obvious herald patch the diagnosis is rarely difficult. *Pityriasis versicolor* may be simulated, but in this condition the lesions are generally confined to the chest and shoulders, the course is extremely chronic and the causative fungus is very easily demonstrable in epidermal scales. *Pityriasis sicca* (streptococcal eczematide) may cause difficulty in children, but the lesions here are often confined to the face (which is seldom spared) and are rarely so diffuse as in typical P.R. Untreated, it lasts longer than P.R. and recurrences are common. *Psoriasis*, except in an acute guttate, punctate or small nummular attack bears little resemblance to P.R. Its lesions are papular and infiltrated and the diagnosis can, if necessary, be established by biopsy. The lesions of chronic *seborrhoeic dermatitis* (*eczématides figurées*) on the chest and back may resemble closely those of P.R. but their distribution, often in the centre of the back and chest, greasy scaling, chronicity and tendency to recurrence are distinguishing features. The seborrhoeic skin is no more susceptible to P.R. than is any other. *Lichen planus* may be simulated very closely by the small papular variety of P.R. but can easily be identified by histological examination. It is interesting to note that cases of lichen planus with a 'herald patch' have been described. There is no suggestion, however, that the two conditions are in any way related. *Tinea corporis* may be confused with vesicular P.R. but the lesions are rarely so numerous as those of P.R. and the fungus can be identified in scrapings from the lesions. Dermatophytids may also resemble the lesions of lichenoid P.R. but the primary fungal infection is usually obvious. *Secondary syphilis* may copy most of the varieties of P.R. and if no other sign of syphilis such as mucosal lesions, adenopathy or an active or healed chancre are present the result of a serum test for syphilis will be necessary to confirm the diagnosis. The *exanthemata*—measles, german measles or varicella may rarely be simulated by P.R. especially in cases with general symptoms.

Points of resemblance between P.R. and pityriasis lichenoides et varioliformis acuta and parapsoriasis guttata will be examined later.

General Symptoms

About 25% of cases complain of a little *itch* in the first week of the secondary rash; a small percentage of patients have severe itch that may last for several weeks and be most intractable. Other signs and symptoms are rare; they include *malaise*, *headache*, *adenopathy*

(glands in the posterior triangle of the neck especially), *low fever*, and fleeting *joint and muscle pains*. Such phenomena occur at the onset of the disease. *Tonsillitis*, *whitlows*, and other *infections* have been noted before the onset of P.R. I have seen one case with a mild *hepatitis* and *icterus* accompanying the secondary rash. When general symptoms occur they resemble those of the milder exanthemata. No characteristic changes in the blood or any other organ have been noted.

Incidence

The case incidence of P.R. varies, according to the American and European observers quoted by Percival,[1] between 3 and 10 per 1,000 patients with skin disease. One author cited, Nekam, gave a much higher incidence of 40 per 1,000. The incidence in the Transvaal among Europeans is about 18 per 1,000 cases with skin disease, and my impression is that the incidence among the Bantu is the same. The figure for Europeans in South Africa is significantly higher than the average in the USA and Europe.

No significant difference in incidence of P.R. in males or females has ever been noted. The disease has been reported from every part of the world and as far as South Africa is concerned it does not seem to have a predilection for any particular race. Niles and Klumpp,[8] however, report only 2 cases in Negroes among 219 American patients of all races. P.R. tends to be more severe and generalized in the Bantu than in Europeans in South Africa.

P.R. is most frequent in patients between 20 and 30 years of age, and occurs almost always between 5 and 50. It is very rare over 50; and the youngest patient I have seen was in his 2nd year.

Most writers state that P.R. occurs most frequently either in spring or autumn or in the colder months of the year. From my records the same seems to be true in South Africa, where the lowest numbers are seen at midsummer in January and February; and there are significantly higher figures in the cold months. Apart from any seasonal changes in incidence there are periods when the disease seems to be epidemic. This is shown in Percival's figures[1] from Edinburgh and has been referred to by many other observers. Such an epidemic in the Transvaal occurred in 1950-52. In spite of its apparently epidemic incidence it is exceptional to find even the most casual relationship between patients. The occurrence of two or more cases within a family or in some institution is rare enough to have warranted publication on a number of occasions.

Recurrences

Second attacks of P.R. are extremely uncommon. In such cases as have been reported the second attack has occurred after some years, e.g. after 4 years in Darier's patient.[9] In the one case I have observed a second attack followed more than 30 years after the first.

Cause

The cause of P.R. is still unknown, but many theories have been put forward. It has been suggested that P.R. is due to infection by a fungus, streptococci, staphylococci, a spirochaete or a virus; that it is a tuberculide, a toxic exanthem due to gastro-intestinal auto-intoxication, or a neurotrophic dermatosis.

The fungus theory had some supporters, notably the late Professor H. Gougerot,[2] who believed the cause of P.R. to be the *Cryptococcus du Boisii* (*C. anomoeon*). This organism can be found in the scales of most cases of P.R. as masses of spores in the follicular sheaths. The dry scales must be mounted direct in xylol and a careful search 'for an hour if necessary' will discover the organisms. This fungus has never been cultured. The majority of writers consider it a saprophyte. (Vidal considered the cause of his *pityriasis circiné et marginé* —now identified with P.R.—to be a similar or identical fungus, which he named *Microsporon anomoeon*). The fact that P.R. affects the scalp frequently in children but rarely in adults is of course reminiscent of the microsporon infections.

Streptococci have been suggested as the cause of P.R. by a number of authors, notably Périn (cited by Gougerot[2]) and Gourvitch,[10] on the basis of finding streptococci in the scales, positive intradermal reactions to streptococcal vaccine, and the appearance of P.R. in patients who were suffering or had recently suffered from manifest streptococcal infections.

The spirochaete theory is considered in an article by Hollström.[11] Following the work of Lennhoff, who was able to demonstrate spirochaetes in specimens of skin stained with mercury sulphide, Hollström cultured spirochaetes from 5 cases, and even managed to subculture them. He claimed that the course of P.R. was markedly shortened by treatment with bismuth or arsenicals. Objections to Hollström's theory are that his bismuth-treated cases recovered as quickly as those treated with the much more potent spirochaeticide arsenic; and that patients under treatment for syphilis may develop typical P.R. (apart entirely from P.R.-like drug reactions). It has recently been suggested by Schirren[12] that the spirochaetes discovered by Lennhoff in P.R. and other skin diseases are simply artefacts of various kinds.

The virus theory is the one most widely held at present. P.R. generally behaves in its course, age incidence and epidemiology, like one of the milder exanthemata and an attack almost always confers life-long immunity.

The portal of entry of the organism, whatever it may be, is also disputed. Contact or droplet infection can obviously be excluded. The wearing of new clothes before the attack, using an affected person's clothes or sharing a sufferer's bed, going to public baths, have all been suggested as factors in a few or many cases. The most plausible idea, to my mind, is that of Louis Brocq who suggested that P.R. might be spread by an insect vector, the flea, and that the herald patch is the site of inoculation. The secondary rash could well result from embolic distribution of the causative organism after its multiplication in the primary lesion; the cutaneous reaction might be one due to infection or to allergic response after the patient had become sensitized. The herald patch is nearly always on the trunk, the site of election for flea bites. The flea theory would fit in with the clothing and public-bath observations mentioned above. Flea-bites are very common in South Africa,

especially among children, for nearly every household has at least one domestic animal. Against the flea theory is the fact that P.R. is commonest in the cold months.

Hissard[13] reports a case where the herald patch appeared a few days after a wasp-sting on the cheek and was followed after 12 days by a typical secondary eruption (but this, of course, cannot be used in support of the flea theory).

Passage

Many experiments in passage of P.R. to animals and man have been made in the past, but no convincing successes have yet been reported. The materials used have been scales and blister fluid (produced by cantharides plasters or CO_2 snow) from herald patch and secondary lesions inoculated by scarification or intradermal injection. Wile,[7] after a long series of failures, produced an eruption in three volunteers by using blister fluid from the herald patch of a case occurring during an 'unusually severe epidemic'. After 3 or 4 days a sparse, itchy, papular eruption appeared, but there was no herald patch at the inoculation site and in no case did the rash last more than a week. Thomson and Cumings[14] had one possible successful transmission with filtered saline extract of scales; and they cite the case of Edelston who, after handling a case of P.R. accidently scratched himself and developed a herald patch at this site and later a typical secondary rash. Gourvitch,[10] and Joyeux, Burnier, and Duché,[15] report unsuccessful trials of passage.

We have made some experiments in passage using ground-up whole skin in a saline-penicillin-streptomycin solution. The fluid was injected intradermally. No reaction whatever was produced and this suggests that the eruptions described by previous experimenters in this field may well have been streptococcides.

If a flea is the vector it is possible that its mouth secretions may contain some substance that facilitates entry of the causative organism into the body; or it might be that the organism must pass a part of its life history in the flea before it can once more cause disease in man. Experiments in which we added ground-up fleas (from a dog) to the above suspension were also unsuccessful.

Attempts to culture a virus on human epithelium and on monkey kidney have so far been fruitless. The possibility of 'silent' growth was excluded by negative results from injection of protein-free washings from the media.

The failure of experiments in passage and in culture does not exclude the possibility of a virus as cause. The culture media used so far may not have been appropriate; and we have not yet tested the possibility that the causative organism may have to multiply or undergo some change in the flea before it is again transmissible to man.

Treatment

The average case requires no treatment beyond reassurance about the prognosis. Over-treatment leads to eczematization of the lesions and increase of symptoms. If itch is severe, one may prescribe baths in solution of potassium permanganate or sodium bicarbonate, calamine lotion with 2% phenol, and aspirin, antihistaminics or even barbiturates for sedation. Sunshine in reasonable doses definitely speeds resolution (the predilection for untanned areas has already been noted). I have used parenteral arsenic and penicillin without the slightest influence on the course of P.R.; and the more recently discovered antibiotics used for virus diseases were equally ineffective. Convalescent serum has been used without significant effect. Cortisone and ACTH had no effect in a few cases tested.

PITYRIASIS ROSEA, PITYRIASIS LICHENOIDES ET VARIOLIFORMIS ACUTA AND PARAPSORIASIS GUTTATA

Pityriasis rosea is mentioned by most authors in discussing the differential diagnosis of two relatively rare conditions parapsoriasis guttata (Para. G.) and pityriasis lichenoides et varioliformis acuta (P.L.V.A.) and it has been tentatively suggested in the past that P.R. and P.L.V.A. might have similar causes. At first sight there would seem little reason to suggest a relationship between P.R. and the other two conditions; but there are some interesting points of resemblance. It should be remembered that there was originally much controversy over the nature of P.L.V.A., which is now recognized as an acute variety of Para. G. for there is little resemblance between the course and appearance of typical examples of the two diseases. Cases representing transition forms between the two, however, present convincing evidence of their relationship.

Parapsoriasis Guttata

The classification of parapsoriasis has undergone many revisions since 1902 when Brocq made his first proposals. Most dermatologists now take the view of Civatte,[16, 17] the greatest authority on the subject, that Para. G. must be dissociated entirely from parapsoriasis lichenoides and *parapsoriasis en plaques*. The last two diseases are related and *formes de passage* link them; and their histological appearances are similar, the important changes being mainly dermal. Both may be forerunners of mycosis fungoides. Para. G. is always a benign disease and the important features in its histological picture are changes in the epidermis.

The rash of Para. G. may quite closely resemble that of P.R. It consists of many isolated lesions, 5-10 mm., slightly papular and pink at first but soon turning to reddish brown, which after a few days are covered by an adherent scale. The infiltrate disappears and there remains only a thick grey scale that can be detached *in toto*. The scale falls off in a week or two leaving a brown macule which fades in its turn. The evolution of a lesion takes 3 weeks but, as new ones are constantly appearing in crops the mixture of lesions at different stages of development gives a quite characteristic appearance. The body and limbs are usually affected, and the hands, feet, face and scalp and mucous membranes are almost always spared. Streamlining of lesions on the back, as in P.R. may be seen. In many cases the disease lasts for years without affecting the general health in the least. Sometimes the rash is recurrent with intervening free spells lasting weeks or months, and the recurrences may be seasonal. There are also typical cases which last only

for a few months and then disappear never to recur. Of 4 cases (adults) seen by me in 1951 2 are still active 4 years later and 2 cleared up within a year. All were Europeans; I have not yet seen Para. G. in the Bantu. Two cases have been seen in 1955, a boy of 10 and a woman of 40.

Apart from the common variety there are also described micropapular, hyperkeratotic, confluent and acute papulo-vesicular varieties. Post-lesional leukoderma of a transient nature is quite frequent and rarely leukoderma is a marked feature (*parapsoriasis en gouttes leucodermique*). Scar formation is not seen in typical Para. G.

The disease commonly begins in youth (Gross[18] quotes Riecke as giving the highest age incidence between 15 and 25 years) and children are often affected. In some cases, among the typical lesions, one sees also some papulo-vesicular and necrotic elements. Such cases represent transition forms between Para. G. and P.L.V.A.

Pityriasis Lichenoides et Varioliformis Acuta

This description is reserved for those cases of parapsoriasis guttata which run an acute course and in which all the lesions are papulo-vesicular or necrotic. The name was coined by Habermann of Bonn in 1925 but credit for discovery of the condition is generally given to Mucha of Vienna (1916). Even before 1916 cases of Para. G. with purpuric and necrotic lesions had been described (cited by Lapière[19]) and the first description of an atypical Para. G. of the P.L.V.A. type (dermatitis psoriasiformis nodularis) is probably that of Moller and Afzelius of Stockholm in 1903 (Gross).

The rash of P.L.V.A. has, according to European and American descriptions, the same distribution as that of Para. G. the body and limbs being affected and the hands, feet, face, scalp and mucous membranes spared. In the South African Bantu, however lesions on the face, hands and feet are frequent (Fig. 3). The lesions appear in crops so that various stages of development can be seen at any given time, and the disease runs its course in a few weeks to a few months or a year at the most. The commonest lesions are red papules and papulo-vesicles, 2-5 mm., which often proceed to central necrosis and crusting to give an appearance suggestive of papulonecrotic tuberculides. Hundreds of lesions are usually present. Purpuric papules, papulo-pustules, bullae and varioliform vesicles are also sometimes present. Typical lesions of Para. G. may also be seen and healing lesions can look identical to those of P.R. In some of my cases in the Bantu the healing stage has copied P.R. so exactly that no other diagnosis would have suggested itself to one who had not seen the primary lesions and examined them histologically. Malaise, fever, joint pains and glandular enlargement may be noted in the early days of the disease; and local infections may precede an attack. Aphthae and ulcers of the buccal mucosa and ulcers or herpetiform lesions of the genital mucosa have been noted, though rarely.

Fig. 3. Pityriasis lichenoides et varioliformis acuta in a Bantu.

Varioliform scarring results in some of many of the lesions and post-lesional leukoderma is common. It can readily be understood from this description that P.L.V.A. was not at first readily accepted as a form of Para. G. Mucha considered it to be so and later descriptions of transition forms showing typical lesions of both acute and chronic types seem to make it clear that we are dealing with two manifestations of the same disease. Cases are described in which the original rash was that of typical chronic Para. G. but where later necrotic lesions appeared and the picture turned to that of P.L.V.A. and the disease ran an acute course.[19] In other cases beginning as P.L.V.A. the picture has changed to that of Para. G. and the course has been chronic.[20, 21]

Recurrences of P.L.V.A. have been described, but the recurrences have nearly always followed closely one upon the other over a space of a few months.[22] P.L.V.A. is a disease of young people with an age distribution like that of P.R. and Para. G. The youngest patient I have seen was between 1 and 2, the oldest about 45. All but one of my South African cases have occurred in the Bantu. It is interesting to note that the incidence of P.L.V.A. is sporadic. The first group (of 13 cases) was seen in Vienna; later outbreaks were reported in England, Poland, Russia, Japan, France and the USA (see table in Gross).[18] Most cases in the literature occurred in spring or autumn (Basset cited by Joulia and le Coulant).[23]

The question of etiology is well reviewed in this recent article by Joulia and le Coulant. It seems most probable that P.L.V.A. is a microbic disease but no micro-organism has yet been demonstrated. These authors refer to unsuccessful experiments by Sirota with a complement fixation reaction and inoculation of guinea pigs and my Mazzaro with culture on allantoic membrane. They note the resemblance of the lesions of P.L.V.A. to those of papulo-necrotic tuberculosis, and refer to successful treatment of the disease with remedies used in tuberculosis. Gougerot's view that benign parapsoriasis is a nodular cutaneous reaction of defence in persons sensitized against a micro-organism seems reasonable. The recurring crops of lesions would correspond with embolism of the causative organisms from a chronic focus to be destroyed in the skin. An intense necrotic reaction (miniature Koch's phenomenon) would be of good prognostic significance. Varying degrees of sensitization in different individuals would explain variations in the clinical picture of parapsoriasis and it could be classified, like Gougerot's trisymptomatic

disease, with the microbic allergides. Gougerot's theory leaves open the question as to whether such a reaction is caused by a specific micro-organism or whether it might be non-specific and result from one of a variety of infective agents. The fact that anti-tuberculous remedies have sometimes been followed by cure is surely no reason to postulate a tuberculous origin for a spontaneously healing disease.

My attention was first drawn to the resemblance between P.R. and P.L.V.A. during the period 1950-52 when, with an 'epidemic' of P.R. in Europeans and Bantu I saw a number of cases of P.L.V.A. in the Bantu. As I have already noted P.L.V.A. in the healing stage in the Bantu can bear a very close clinical resemblance to P.R. In the literature we find many references to cases in which differential diagnosis of the two conditions presented the greatest difficulty. I found that I was not the first to note a close resemblance between the two conditions. Drake,[24] in 1930, discussing cases of P.L.V.A. shown at the Royal Society of Medicine, London, remarked that most of them exhibited the peculiar streamline distribution along the lines of the ribs and in the axillary folds, and the greasy mica-like scale was also like that seen in P.R. Hence it was suggested that they had a similar cause. Macleod[25] also noted that the distribution of lesions resembled that of P.R. and suggested the possibility that it might be due to a specific infective virus.

I was distracted from pursuing the subject at the time by Dr. A. Civatte whom I consulted over the histological relationships. He found difficulties in admitting a relationship as the epidermal lesions in P.R. (eczematide) are not comparable to those seen in P.L.V.A. or Para. G. However, I returned to the inquiry recently when, with a rising incidence of P.R. I saw a case of P.L.V.A. and a case of Para. G. within one week. (I have since seen 3 more cases of P.L.V.A. and a second Para. G.) On checking my records I discovered a fact that had not previously struck me; the 4 cases of Para. G. which I had previously seen in South Africa all first consulted me during the period 1950-52. It is interesting to note that Drake,[26] in 1932, referred to the sporadic incidence of cases of Para. G.

There are many points of similarity in course and clinical appearances between P.R. and P.L.V.A.; the similarity is less marked in comparing P.R. and Para. G. I have summarized these points in Table II.

Histology

The important changes in Para. G.[17] are those in the epidermis. There is a dermal infiltrate, almost exclusively lymphocytes, which may be a prominent feature in the early stages. This infiltrate is diffusely distributed in the papillary layer and even a little deeper. Whatever the quantity of infiltrate in the dermis it is always abundant in the epidermis which it invades from the start in all its thickness. It penetrates immediately to the superficial layers seemingly without any resistance. This exocytosis is not preceeded by exoserosis as is the case in eczema where spongiosis makes a bed for the infiltrate. The attack on the epidermis is at first in multiple tiny foci. Wedges of large monocytes make the initial penetration and are quickly followed by lymphocytes (Fig. 4). The Malpighian cells lose their connecting filaments, swell and separate to give place to the invading cells. The epidermis soon repairs itself and looks again relatively intact but powdered with lymphocytes. It remains, however, for a time incapable of forming keratohyaline and dries off in parakeratosis to form a scale at the expense of its upper layers.

Fig. 4. Parapsoriasis guttata. A wedge-shaped focus of monocytes and lymphocytes penetrates the epidermis; parakeratosis and dermal infiltrate.

Sometimes the epidermal alterations are still more marked. As well as the dislocation from exocytosis there may also be degeneration. The corpus mucosum is sown with eosinophilic hyaline bodies, the remains of Malpighian or invading cells. This touches on the picture seen in P.L.V.A.

In P.L.V.A.[17, 27] the epidermal alterations are even more marked. There is an abundant exoserosis and even vesicular cavities between the Malpighian cells. These cells enlarge, their cytoplasm becomes transparent, their nuclei alter and their outlines are ill-defined. The whole corpus mucosum may become homogenized and undergo hyaline degeneration so that the central areas are

only a disorganized mass, a cone of necrosis with its summit in the dermis (Fig. 5). The parakeratotic scale which originally covered the lesions is shed and replaced by a serous crust. Below the necrotic area is a dense mass of lymphocytes, disintegrating erythrocytes and swollen cells resembling fibroblasts. The line of demarcation

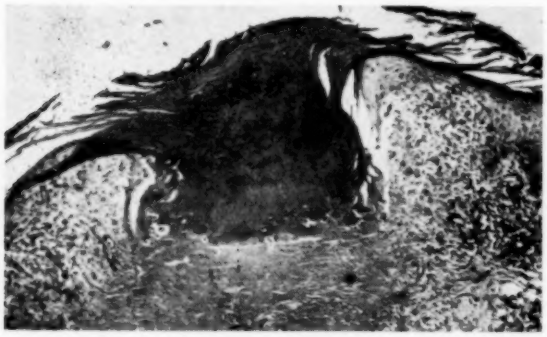

Fig. 5. Pityriasis lichenoides et varioliformis acuta. A disorganized mass of hyaline necrotic tissue is present. The line of demarcation between epidermis and dermis is obliterated. Diffuse cellular infiltrate in upper corium.

between epidermis and dermis is wiped out. There is a marked perivascular and diffuse infiltrate in the upper corium. The walls of the arterioles are thickened and infiltrated, their lumina narrowed. The vascular changes do not, in the majority of cases, suggest that this is primarily a necrosis due to vasculitis. Touraine *et al.*,[28] however, describe one case where vascular changes were so marked that they could be suspected as the primary lesion, and Duperrat,[29] like Gougerot, very tentatively classifies P.L.V.A. as a necrotic dermal allergide. For most observers the epidermal process seems to be primary.

As Civatte remarks, the histological changes in P.R. are not strictly comparable to those in P.L.V.A. described above. The process is probably primarily epidermal (this is disputed), but the first change is inter- and intracellular oedema going on to spongiosis, and the formation of irregular and ill-defined vesicles (Fig. 6). These are invaded by medium or large mononuclears and may also contain epidermal cells. The stratum granulosum disappears and parakeratotic scales form. When visible vesicles form they are found to be subcorneal and even in a severe vesicular and crusting case there is no epidermal cellular degeneration comparable to that seen in P.L.V.A. There is capillary dilatation in the upper corium and a pericapillary round cell infiltrate. This infiltrate can be very dense and widespread in lichen planus-like P.R.

In my own cases the histological changes have, on the whole, conformed to the classical descriptions given above. But it is not always possible to distinguish Para. G. and P.R. from the histological picture. One case showed features such as may be encountered in both diseases. The patient was a boy of 10 who had suffered for 9 months from a rash on the trunk consisting of P.R.-like lesions and a few scattered small papules of

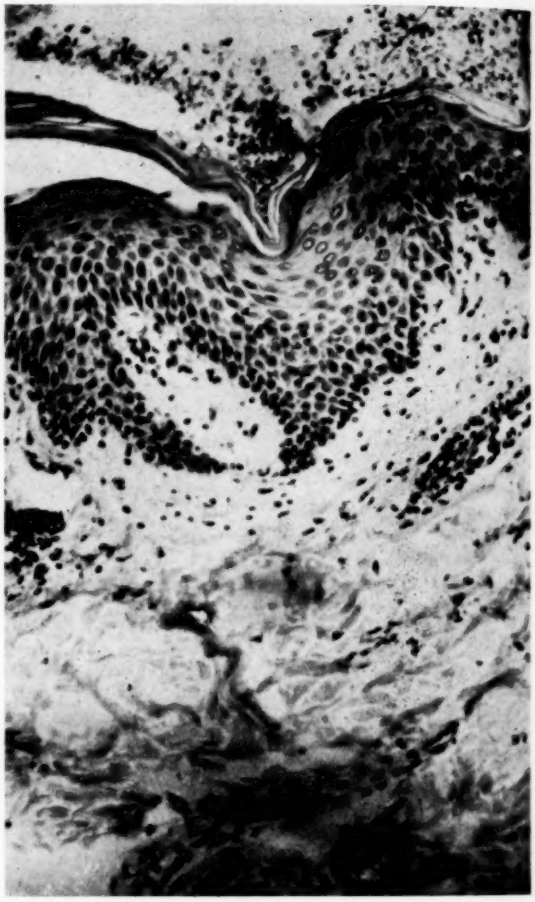

Fig. 6. Pityriasis rosea. The epidermis shows inter- and intracellular oedema and parakeratosis. Part of a subcorneal vesicle is visible. Perivascular infiltrate in the corium.

the Para. G. type. Histological examination of a papule showed one focus where the Malpighian cells had lost their filaments and the epidermis was infiltrated throughout its thickness by monocytes and lymphocytes. This area was covered by a parakeratotic scale. In the adjacent epidermis were scattered monocytes and lymphocytes together with patchy spongiosis. Such a picture is suggestive of Para. G. In another area the spongiosis had progressed to a small irregular cavity containing a few lymphocytes, monocytes and epithelial cells. It was subgranular and suggested an early attempt at vesicle formation as is seen in P.R. The upper corium showed a moderate infiltrate of monocytes and lymphocytes. The eruption was still present 6 months later, the papular lesions predominating, and it seemed certain that the case was one of Para. G.

CONCLUSIONS

Seasonal and epidemic variations in the incidence of P.R. are admitted by the majority of observers; the

sporadic appearance of cases of P.L.V.A. is striking and it has been noted by Drake that cases of Para. G. also seem to come in crops. In my own experience cases of P.L.V.A. and Para. G. have been seen on two occasions when P.R. was epidemic. This suggested a relationship between P.R. and the two other clinically dissimilar but actually identical diseases. I have asked colleagues in Britain and Europe to refer to their records to see whether any such relationship in incidence could be traced, but in the (short) periods they could survey, nothing significant was discovered. It is interesting to note that 8 cases of P.L.V.A. were seen in London during the period 1926-29 (Gross); and Percival writes: 'During the period 1924-1930 the case incidence of P.R. increased considerably, reaching its highest point in 1930 and the increase appears to be maintained during January-June of 1931. It would seem, therefore, that since 1925 Edinburgh has had one "epidemic" of P.R. and is at present experiencing a second'.

Further study shows that there are many points of resemblance in the clinical appearances, age incidence, seasonal incidence, course and immunological behaviour, between P.R. and P.L.V.A. and to a less marked degree between P.R. and Para. G. It seems not entirely unreasonable to suggest that these conditions may have a common, probably viral, cause or be due to closely related infective agents. There are naturally objections to the theory. Why is P.R. common and always with us and the others rare and, in the case of P.L.V.A. certainly, sporadic in incidence? How can we postulate a common cause for two acute conditions and one chronic condition? The reason could lie in variation in virulence of the causative organism or in variation in immunological response to infection in the host. It is possible that the consistent failure of attempts at culture and passage is due to the fact that the lesions are allergides and that the microbic allergen is, therefore, being destroyed in the skin.

There are definite differences between the histological changes seen in the 3 conditions and it is generally easy to differentiate P.R. from the others. This, too, might be explicable by variations in reaction to a single cause. There is precedent for the suggestion and one can offer the examples of syphilis, tuberculosis and leprosy where the histological pictures produced at different stages can differ vastly.

SUMMARY

The clinical picture of pityriasis rosea in its typical and atypical forms is fully reviewed. Some variations in the behaviour of the disease in the Transvaal, particularly in the Bantu, are described. Pityriasis rosea seems to be commoner in the Transvaal than it is in Europe and America. Theories of origin are discussed and experiments in culture and passage are described.

It is noted that sporadic outbreaks of pityriasis lichenoides et varioliformis acuta and parapsoriasis guttata have coincided with epidemics of pityriasis rosea in the Transvaal. The clinical appearance, course, epidemiology etc., of the three diseases are discussed and compared and it is suggested that they may have a common cause or closely related causal agents.

TABLE I. THE VARIETIES OF PITYRIASIS ROSEA

Macular

Punctate, Guttate, Nummular, Circinate	Bilateral	Solitary plaque
	Unilateral	Cervico-cephalic
	Localized	Scalp
	Generalized	Bathing-trunk distribution
	Confluent	P.R. *gigantea*
	Diffuse	*P. circiné et marginé.*

Urticarial

P.R. ortié (Hallopeau), P.R. urticata (Vorner)

Papular

Maculo-papular
Follicular
Miliary
Lichen-planus-like

Vesicular

Maculo-vesicular
Papulo-vesicular
Varicelliform

Pustular

Pityriasis Rosea et Pustulo-crustosa

Other Atyrical or Complicated Types

Psoriasisform
Eczematous
Lichenified
Erythematous
Non-squamous
Haemorrhagic
Chronic (P.R. perstans)
With post-lesional hyperpigmentation or depigmentation

TABLE II

	Pityriasis Rosea	*Pityriasis Lichenoides et Varioliformis Acuta*	*Parapsoriasis Guttata*
Herald Patch	A feature of the disease, occurring in 12-90% of cases according to various authors.	Solitary lesion preceding main eruption has been described.	Not noted.
Rash	Commonly maculo— or papulo-squamous lesions, but many variants described. Lesions appear in crops.	Papules or papulo-vesicles going on to necrosis. P.R.—like lesions also common. Lesions appear in crops.	Papulo-squamous lesions. Lesions appear in crops.
Distribution	Body and limbs. Face, scalp, hands, feet and mucous membranes usually spared in adult Europeans. Face and scalp often affected in Bantu at all ages. Streamline distribution over ribs on back.	As for P.R.	Body and limbs. Face scalp extremities and mucous membranes usually spared. Streamline distribution on back.
General Symptoms	Malaise, headache, low fever, adenopathy, fleeting pains, may occur in early stages	As for P.R.	Nil.
Incidence	Relatively common.	Rare.	Rare.
Duration	Average case 6–12 weeks. Limit about 1 year.	6 weeks to 6 months. Rarely as long as 1 year.	Often very chronic, persisting with or without remissions for many years. Cases with typical lesions but running short non-recurrent course are described.

TABLE II (CONTD.)

	Pityriasis Rosea	*Pityriasis Lichenoides et Varioliformis Acuta*	*Parapsoriasis Guttata*
Recurrences	An attack usually confers lifelong immunity. Very few cases of second attacks described; intervals between attacks in such cases usually several years at least.	Recurrences or second attacks after long intervals very rare. Recurrences over short period described; probably all part of a single attack.	Regularly recurrent attacks a feature of some cases. Recurrence after long period of freedom is rare.
Age at Onset	Young people. Highest incidence between 20 and 30	Young people 15–25.	Young people 15–25
Seasonal Distribution	Commonest in the colder months.	As for P.R.	Not applicable.
Epidemics	Epidemics', apart from seasonal fluctuations, noted by many observers. Increased number of cases seen in Transvaal 1950–52 and again in 1955.	Cases have occurred in sporadic outbreaks in various parts of the world since the first cases were described in Vienna. 8 cases seen in Transvaal 1950–52, 4 in 1955.	Sporadic occurence of cases mentioned by Drake. 4 cases seen in Transvaal 1950–52, 2 in 1955.

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DISTRIBUTION OF INTERNS *

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In the post-war years the world entered the era of shortages. Wherever one turns one finds that the demand far exceeds the supply. This applies not only to raw materials but also to man-power, the shortage of which is evident everywhere. The employee can therefore dictate the conditions of employment to the employer. The demand for interns also exceeds the supply, and the intern today is in a position to dictate the conditions under which he will work. At some hospitals this dictator-like attitude has only been overcome with great difficulty.

The distribution of interns is so closely inter-related with the shortage of interns, that one has included the causes of the shortage of interns in this paper.

The shortage of interns may not be felt in teaching hospitals and other large general hospitals, but it is evident in the smaller hospitals and the platteland hospitals. It is of vital importance to the smooth administration of a hospital, for the intern forms the backbone of the full-time staff, and a shortage of interns directly affects the medical care of the patient. This shortage has become so acute in some hospitals that drastic steps will have to be taken to relieve it, and will have to be taken immediately.

* A paper presented at the South African Medical Congress, Pretoria, October 1955.

CAUSES OF THE SHORTAGE IN INTERNS

It is only since compulsory internship was introduced that the shortage of interns has arisen. In the days of voluntary internship the hospitals were well staffed although housemen were paid much lower wages. At that time there were not so many hospitals as today, but on the other hand the medical schools have increased since those days and are producing more medical graduates. In my opinion the present shortage of interns is not a true shortage, but only an apparent shortage, which was created in the post-war years. To estimate the true shortage of interns one must first remove the causes of this pseudo-shortage, which are as follows:

(*a*) Directly after the cessation of World War II a number of ex-volunteers applied to the Cape Town and Witwatersrand medical schools for admission as medical students. These medical schools already had their

normal quota of students, and with the concurrence of the South African Medical and Dental Council they raised the student quota so that the ex-volunteers could be accepted. When those students graduated, it was found that the number of vacant posts available were not sufficient for the new graduates, and the Medical Council sent out an appeal to the provincial authorities asking them to create extra posts for interns. This was done and everybody was happy; interns were given fewer patients to look after and shorter hours to work, and posts were created in specialistic departments. Hospitals, though they still had the same number of patients as previously, had more intern posts to fill.

Once the ex-volunteers had all qualified, the medical schools reverted to their normal quota of students. Although at about this time the Pretoria medical school's first final-year students graduated, their number was less than that of the ex-volunteers of the previous years. The Medical Council never asked the provincial authorities to withdraw the posts that were created to cope with the ex-volunteer graduates, and it is these posts that are today causing a pseudo-shortage of interns.

It is interesting to note the total number of recognized intern posts in the Union in the past few years. The figures do not include posts in mental, infectious-disease or tuberculosis hospitals, but only recognized general hospitals in the Union.

Year	*Posts*	*Interns Qualified*	*Shortage*
1952	477	392	85
1953	477	294	183
1954	477	321	156

From these figures one can see that the shortage of interns will be with us for many years to come if the allocation of intern posts to recognized hospitals is not revised. In the years 1950, 1951 and 1952 there were respectively 488, 437 and 461 final-year students in the medical schools. Even if all these students passed they would not have been sufficient for the available intern posts. One must not forget that many interns do their internship in the Rhodesias or overseas, and that therefore the final number available for the Union hospitals is actually less than the figures quoted.

(*b*) Another cause of the pseudo-shortage of interns is that many posts classified as posts for interns are in departments of a highly specialistic nature, nearly all in teaching hospitals. These should not be allowed, for they are contrary to the conditions laid down by the Medical Council in their original regulations governing the year of internship. Interns should only be allowed to be employed in the departments of general surgery, medicine, gynaecology, obstetrics, and paediatrics. Other specialistic departments should make use of senior housemen or registrars. No intern should be allowed to do his internship in a mental, infectious-disease or tuberculosis hospital. When interns are allocated to these posts one is creating a pseudo-shortage of interns. It was never intended by the Medical Council that interns should do their year of internship in posts such as these.

In my opinion the intern question is so important at present that the South Africa Medical and Dental Council should appoint a commission to investigate the whole question of allocation of intern posts to recognized hospitals.

Such a committee should investigate how many indigent hospital patients an intern should be responsible for, his medical duties, his hours of work, and the conditions of his employment. These should be uniform throughout the Union. In my opinion the old idea of allocating fewer patients to an intern in a teaching hospital is incorrect. A patient needs the same attention no matter where he is hospitalized. The intern attending him is still required by the Medical Council to write the same history and follow-up notes. The intern in a teaching hospital has the advantage that he has registrars and senior housemen to help him; if there is to be a difference, the number of patients should be fewer in the other hospitals. The Medical Council never intended that the conditions of internship should be different in different hospitals, and therefore the allocation of posts in hospitals should be uniform.

I am convinced that with a re-allocation a large number of intern posts could be done away with. One example may be quoted to stress this point. In June 1954, when new appointments were made, two interns at a certain Reef hospital discovered that the hospital would be short of 2 interns out of a total of 11. They promptly resigned, and gave as their reason that with a shortage of 2 interns they would have to work too hard. They were applying for posts in a large hospital in Natal, which would be fully-staffed. Within 2 months of going to Natal, they were back on the Reef again, stating that they could not 'stick' it at the Natal hospital, because there was no work for them to do, as the interns were literally falling over one another.

From the above it is evidently impossible to estimate the true shortage of interns in the Union under the present system of allocation of interns to hospitals.

METHODS OF DISTRIBUTION OF INTERNS

The present method of distribution whereby interns apply to hospitals for posts does not result in an equitable distribution. The teaching hospitals and the larger city hospitals have a definite advantage over the smaller hospitals, and this usually results in the former being full-staffed and the latter, including the Reef hospitals, being under-staffed, or completely without staff.

At present a prospective intern applies to as many hospitals as he wishes and it often happens that he accepts more than one post. When the examination results are made known, he accepts the hospital he prefers, and at the same time informs the other hospitals that he cannot come. This causes great inconvenience and disruption in many hospitals.

The present method of distribution of interns should be scrapped and the distribution of the interns taken over on the following lines:

The South African Medical and Dental Council should nominate a central commission who will be responsible for the equitable distribution of interns to hospitals for each year. Interns must send their applications for posts to this commission and must nominate 3 hospitals in which they wish to work, in their order

of preference. Three are necessary, because a hospital nominated by the intern may be fully-staffed and then the commission will appoint the intern to the hospital of his second or third choice.

The commission would obtain the number of probable passes from each medical school, and then allocate the available interns on a pro-rata basis to hospitals. It should not be difficult to estimate the number of passes because the percentage of annual passes does not vary greatly. This scheme would still give the intern a free choice of hospitals, and would not cause inconvenience to anyone. An intern should not be allowed to change from one hospital to another till he has completed 6 months. Interns should commence duties at a fixed date (or within a week of it) in all hospitals in the Union.

Interns should be allowed to complete their year of compulsory internship in a non-teaching hospital, and only a maximum of 6 months in a teaching hospital. A regulation of this nature will not be acceptable to all, but it is in keeping with the reasons that moved the Medical Council to legislate for a year of compulsory internship.

The year of compulsory internship was instituted so that the inexperienced, newly-qualified doctor before practising on his own would obtain a year of practical experience and training under supervision, and would learn the fundamental principles of general practice. The great majority of interns will be general practitioners after their year of internship and it is essential that they should be thoroughly equipped for general practice. In the teaching hospital the medical staff is completely or almost completely composed of specialists, and there the intern seldom if ever sees a general practitioner or is taught by one. How is it then possible to teach the intern the art of general practice? The type of patient admitted to a teaching hospital is generally not the type of patient found in general practice. The patients in teaching hospitals suffer mostly from complicated conditions requiring numerous investigations before a diagnosis can be made. Operations performed are usually of an advanced and complicated type and would never be done by the general practitioner. The minor operation performed by the general practitioner is a rarity in the operating theatres of the teaching schools.

Further, in a teaching school a number of senior medical staff are attached to each medical or surgical 'firm', and the most junior of these is the intern. How will he ever learn to accept responsibility with so many senior members? The teaching hospitals have a specific purpose in the training of medical men, but they should train medical men who want to specialize or obtain more experience in particular branches of medicine. Let the intern do his year of training in a hospital where he will benefit most and in a hospital where the principles laid down by the Medical Council for the training of an intern pertain.

At present the conditions of service of interns vary from hospital to hospital, and this in my opinion is not desirable. All hospitals should have uniform conditions of service laid down by the Medical Council. The Medical Council should appoint medical inspectors to visit the hospitals and inspect the work carried out by interns and report to the Medical Council whether a hospital is still suitable as an intern training centre. Interns who are found to be doing work below the standard required by the Medical Council should be penalized by the Council and ordered to do an extra term of internship. Interns who are charged with misconduct or other irregularities should immediately have their work investigated by an inspector of interns and, if found guilty, punished by the Medical Council.

Today there is no way in which an intern can be punished. Cases reported to the Medical Council are not even reprimanded. All that happens is that the unfortunate medical superintendent who is brave enough to report an intern will find that his hospital is boycotted by interns and that he has no interns. Interns must be made to realize that the year of internship was instituted for a definite purpose.

Many of the difficulties and problems outlined in this paper may not exist in teaching hospitals, but they are a real problem in most hospitals.

In conclusion, I would express the opinion that the South African Medical and Dental Council made a mistake in naming the extra year of practical study a year of compulsory internship. The medical man by his very training is an individualist, who has been taught to think and act for himself, and the word compulsory immediately antagonizes him. The Council should change the curriculum. The medical student should do 7 years of study before he is qualified. The 1st year should be a pre-medical year of study, in a recognized University. Next the student should study for 5 years at a medical school and during that time he should qualify in all the theoretical and certain practical aspects of medicine. The 7th year of study should be a year of practical experience in recognized hospitals in the Union. The student should obtain his degree and the right to practice medicine only after he has completed and passed the 7 years of study. Let the student realize he is a student till the end of his 7th year.

ABSTRACT : UITTREKSEL

Syphilis in Industry. John G. Downing (1955). J. Amer. Women's Assoc., **10**, 379.

Since the advent of penicillin therapy the incidence of syphilis has greatly decreased in the United States. As a result an attitude of complacency towards the disease has set in. It is estimated that there is still a residue of 2 million persons requiring antisyphilitic treatment in the United States, and that in 1953 about 91,000 civilians acquired the disease. That the battle is by no means over is shown by the fact that in 1953 in 18 states an increase in infectious syphilis was reported.

The author wrote to a large number of medical practitioners in industrial plants. The latter reported that the present incidence in industry of the disease was 1% or about half of what it had been 10 years previously. The cost of finding even these few cases is still worth while to industry. There is still need to educate industrialists regarding treatment of those employees found with positive blood tests for syphilis. It is strongly urged that the question of their being employed should be decided by competent physicians; otherwise gross injustice may be done. A reduction of control efforts by the public health authorities is not only short-sighted but foolhardy.

F.W.F.P.

CORRECTIVE SURGICAL PLANING

A NEW DERMABRASIVE TECHNIQUE FOR THE REMOVAL OF SCARRING AND OTHER LESIONS*

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Despite progress made in the understanding and handling of other aspects of the disease, there was until recently no satisfactory approach to the problem of acneiform scarring. The first forward steps came from Kroymayer,[1] Schreus[2] and Iverson,[3] who introduced and developed various techniques, using grinding wheels and sandpaper. A further and greater advance took place in 1953, when Kurtin[4] described his dermabrasion operation. His method, using ethyl-chloride refrigeration and a stainless-steel brush, powered by a dental motor, has acquired rapid popularity throughout America, and a variety of minor modifications have been added which, it has been claimed, render it a reasonable means of overcoming a hitherto permanent disability. In the United States some exceptional results have been reported. If my own are less spectacular it may be because I have had fewer cases and, in consequence, have not yet mastered a difficult technique. Certainly in my earlier patients I did not carry the abrasions to anything like the depth at which I now work.

In dealing with these cases, certain problems recur constantly. It is, therefore, proposed to discuss them in the order in which they are commonly encountered.

The first, and by no means the least, problem is the choice of case. It is relatively simple to say that the operation is designed for the burnt-out, inactive case, but it is much more difficult to decide just when this stage has been reached. When one observes the patients closely, one finds that many cases which one previously would have classed as 'well', in the sense that they no longer had active acne, are actually still developing occasional pustules. It is these pustules which constitute the problem. Eller[5] has suggested that an operation at this stage is not contra-indicated and that if it is done there is a tendency for further pustules to avoid the planed areas. In one of my cases this has certainly occurred; in another, the position is quite the reverse. In every case in which the smallest pustule or cyst has been opened, either deliberately or accidentally, during the operation, impetiginization of the treated surface has followed. I feel now that one must either reject these cases entirely or carefully avoid any pustules that are present, and this I have endeavoured to do in the last few cases of the series.

Assuming that the case is burnt out, what degree of improvement can be promised and in what time? This depends on several factors; in particular the type of scarring present, its location, the texture of the skin, the anaesthetic used and the technique of the operator. Contrary to expectation, I should say that the greatest improvement can be expected in patients with elevated scars. This is because one can work on until one is sure they are flat and, at the same time, maintain one's bearings *vis-a-vis* the surrounding skin. With a depressed scar one can remove the scar only to replace it with an equally unattractive gutter or saucer-like groove, the size of which is determined by the size of the brush employed; or one can attempt to avoid this by working more lightly only to find that the borders of a scar which were no longer apparent after planing and whilst the patient was on the table are only too apparent after healing and separation of the crusts. I would say, categorically, that one must go as deep as seems necessary and then a little deeper still. Up till now, no one has reported failure of the epidermis to regenerate, no matter how deep the planing; and, although work at this level is often attended by considerable bleeding, this can be controlled by pressure from swabs moistened with adrenaline or, in extreme cases, with Oxycel.

In all areas, but particularly on the neck, close shaving and careful preparation are essential. I have only treated 2 cases with lesions in the neck area and both gave trouble from secondary infection. On the other hand, as much of the scarring was of the hypertrophic type, the end-result was cosmetically very satisfactory.

The next problem is the anaesthetic. Most if not all of the reported American planings, have been done under ethyl-chloride refrigeration, following pre-chilling of the face with polyethylene-glycol face-packs. In this method, the pack is cooled in the freezing compartment of a refrigerator and is then covered with a thin towel and given to the patient, some 20 or 30 minutes before the operation, to hold in contact with the area to be treated. On the table this area is frozen, one section at a time, by a coarse ethyl-chloride spray. Evaporation of the spray is assisted by a jet of air from a motor-driven blower. It is important that the air jet should fall on the skin some inches below, and not at the same point as, the spray of ethyl-chloride. From this position, the air sweeps up over the affected surface but, if it impinges on the same point as the ethyl-chloride, the latter is dissipated and rendered ineffective. My earlier cases were treated in this way, but since then I have used general anaesthesia. I have done so because my patients complained that the operation was painful and in consequence they were unable to keep still. As one often has to work close to the eye, this is a matter of some importance. Furthermore, I found it difficult to achieve the right depth of freezing. Either my freezing was too superficial and had worn off before the planing of an area was complete, or it was too deep and obscured the outline of the scars, as it clung like hoarfrost to the facial hairs. Not even the introduction of Freon as an

* A paper presented at the South African Medical Congress, Pretoria, October 1955.

alternative to ethyl-chloride has tempted me to change back. Freon has certain advantages over ethyl-chloride in that its action is very rapid and more intense, but it is also more painful and more destructive, and the erythema following its use tends to be more persistent. Erythema is one of the major complications of the operation and anything that augments it should, if possible, be avoided.[6]

For general anaesthesia I have used Pentothal, gas and oxygen. Ether and other inflammable gases and osygen. Ether and other inflammable gases must be aboided because the apparatus is not sparkproof. With a general anaesthetic there is, of course, no pain and one has as much time as is required to abrade, and abrade again, the affected areas. There is no crowding of an already restricted field by a spray, a sprayer and a blower, and their absence reduces the chance of infection. Bleeding, however, is freer than with refrigeration and is a problem that I have not yet overcome. Its effect in obscuring the field can be minimized by careful positioning of the patient so that the areas planed first remain lowermost. This ensures that the blood oozing from them runs away from and not over untreated zones. It is of importance not to try and control the bleeding by swabbing. The rotating brush has an uncanny way of picking up the merest thread of cotton and when this happens, the remainder of the swab is drawn into and wound round the brush, arresting and often breaking its somewhat delicate mechanism. The skin is softer than when frozen and the brush tends to skid over its surface. This can be countered by steadying the working wrist with the other hand and by keeping the skin on stretch with small hooked retractors. Without the blower, there is more tendency for the blood and the abraded particles to be thrown up over the operator, so that an apron and a plastic mask must be worn and an assistant must be on hand to clean the mask with a wet towel, for the thin film of blood thrown on its surface clots very rapidly. Recently, at the instigation of Dr. Lombard, the anaesthetist, I have developed a guard which fits closely around the brush head to prevent spattering and yet, at the same time, allows a view of the field. It has functioned so well in the last two operations that no protective clothing has been necessary. Intubation is, of course, necessary, for an anaesthetic face-mask would cover the areas to be treated. It is therefore wise, before the operation, to discuss with the anaesthetist the direction in which he plans to bring out his tube, in order that it can be placed so as to interfere as little as possible with the subsequent procedure.

In the operation itself the brush is carried downwards with short superficial strokes barely touching the surface, with much the same motion as that used in shaving. On the cheeks, the forehead and the chin I prefer to use the broader so-called No. 3 brush, and only around the glabella and in the nasal and retro-auricular folds do I use the finer No. 1 brush. The replacement of these brushes is a matter of some difficulty, for they wear down with surprising rapidity, and are not obtainable in South Africa. I have not yet found out what happens to the wire strands as they disappear. It is to be hoped they are not imbedding themselves in the skin to give rise, at some later date, to a crop of granulomata. If the scarring is in a restricted area, then its borders should be abraded only lightly in an effort to prevent guttering and to shade it into the surrounding tissues. No fear should be felt if the brush has to be carried many times over the affected area. If the bottom of the scar can still be seen, then it follows that there must be epidermis remaining which will regenerate and cover the treated surface. I have not found that painting with gentian violet or with any other coloured antiseptic helps materially in the recognition of the scars.

The operation over, the next question is that of dressings. On the table, the treated area is covered with Telfa strip. Telfa is an inert hydrophobic plastic film, perforated by pores large enough to pass the exudate of abraded surfaces, but small enough to exclude granulation buds. It is bonded, on its reverse side, to Webril non-woven cotton fibre, which is highly absorbent and draws blood through the perforations. The dressing is non-adherent and strips painlessly from the treated surface. It is therefore an improvement on the dry sterile gauze, the Xeroform gauze and the penicillin *tulle gras* which were at first advised. I have not yet determined the ideal moment for its removal. In theory, it should remain on until the 6th or 7th day, when it and the crusts should be removed together. In practice, because it has to be built up from numerous fairly small pieces cut to fit the contours of the face, it will not remain in place this length of time, and I have usually removed it some 4 hours after the operation. By this time, fairly firm crusts have formed and I feel that when these are allowed to dry, a process that can be assisted by a current of air from the blower, no further dressing is necessary. When this has been done, the result has been satisfactory. However, most patients feel that they would like some covering over the area and in these cases I have used masks of sterilized lint with eye, nose and mouth holes, applied smooth side to the skin and kept in place with ties. These however, are unsatisfactory. They tend to stick to any area still oozing and later, during sleep, the crust may be separated at its edges by movements of the mask. Infection then follows and spreads rapidly. The last 3 cases, despite some discomfort, have kept their dressings on for 48 hours. This has given a good result and the crusts have stayed firm and uninfected. The crusts fall off spontaneously on the 6th or 7th day. If not, they can be washed off with soap and water or lifted from the skin with sterile forceps.

Of the several complications hitherto described, I have seen but 3—persistent erythema, hyperpigmentation and milia. An erythema is present in every case when the crusts come off, but disappears in the course of 4 or 5 weeks. Occasionally, it lasts twice as long. Perhaps I am wrong in expecting it to go sooner and should record the normal period as somewhere between these two extremes. My cases have been treated during the winter months, and therefore I cannot comment on the possible effects of the sun, but cold winds and the use of soap and water and good-quality cosmetics do not seem to be deleterious. Pigmentation is a more serious problem. It appears 2 or 3 weeks after the operation and once present, may linger for months. Strictly confined to the abraded areas, it is composed of uniform

light-brown sheets of pigment without reticulation or telangiectases. A biopsy from one of these areas has not, so far, been possible, and therefore the location of the pigment remains uncertain. My feeling is that it constitutes an attempt at protection of the newly-formed attenuated tissues. It is not necessarily most marked in those patients who have been the most heavily planed; in fact, one of the patients with pigmentation had probably the lightest planing of any in this series. The milia are usually numerous but very tiny and superficial. They often appear as long as 2 months after the operation, but are easily removed by slitting the skin over their surface and then pressing out their contents with a blackhead remover. Persistent irritability, mentioned by Kurtin,[4] and the eczematous reactions described by Edelstein[7] I have not so far encountered. Pyoderma, I have mentioned when speaking of dressings.

It is still too early to say if any of these sequelae will be permanent, but the nature of the operation suggests that this is unlikely.

For what other conditions is the planing procedure indicated? In addition to scarring, Kurtin[4] has reported improvement following its use for the removal of superficial epitheliomata, keratoses, tattoo marks, wrinkles, keloids, adenoma sebaceum, and lichenification. Eller and Rein have used it to treat cases of fixed freckles, senile lentigines, chloasma, lupus erythematosus, and xeroderma pigmentosum. In South Africa it may come to have a place in the treatment of these keratotic hands which are seen so often in farmers and in which one hesitates to use radiotherapy because of the multiplicity of the lesions and the proximity of underlying tendons. Diathermy, trichloracetic acid and currettage may remove the lesions, but their cosmetic effect is patchy and, therefore, undesirable and the more uniform appearance of a planed surface should prove preferable.

I have not attempted to tabulate or to draw statistical conclusions from my results. The numbers—15 cases, of which 4 have been planed twice, and one 3 times—are too few and the time that has elapsed since their treatment is too short. I can, however, say that in every case the patient's appearance has been improved, several times substantially, and sometimes even to the point where both I and the patients have felt satisfied. Granted that the procedure and the technique are still in their infancy and that its complications are still mostly unknown, it does seem to constitute an advance, and I feel that the future will see a widening of its scope and the establishment of its acceptance.

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THE SOUTH AFRICAN NATIONAL COUNCIL ON ALCOHOLISM

Representatives of Societies on Alcoholism, drawn from as far afield as Cape Town and Durban, with observers from Kitwe in Northern Rhodesia, met at a conference at the Witwatersrand University, Johannesburg, under the chairmanship of Dr. R. A. Mathews on 3-5 February, 1956. The main object of the conference was the establishment of a South African National Council on Alcoholism.

A constitution was adopted and it was agreed to make application for registration as a Welfare Organization. The objects of the council are:

(*a*) To deal with all matters of a national character appertaining to alcoholism and also with all matters of an international character appertaining to alcoholism in so far as they relate in any way to the concerns of the area served by the Council.

(*b*) To increase public understanding of alcoholism, its nature and treatment.

(*c*) To make this knowledge effectual in an attempt to deal with the problems of alcoholism.

(*d*) To promote the tenets that the alcoholic is a sick person, that the alcoholic can and should be helped and that alcoholism is a public-health and social problem and therefore a public responsibility.

(*e*) To affiliate with any national body having similar objects.

There was unanimity amongst delegates that the strength of the National Council will lie in the constituent societies throughout the Union. These societies are concerned purely with the management of alcoholism and will not engage in activities designed to promote or prevent the consumption or sale of alcoholic beverages.

In order to promote its objects the Council will in collaboration with constituent bodies develop a programme for:

(*a*) Educating the community in regard to alcoholism.

(*b*) Establishing information centres on alcoholism where suffereres and relatives may seek information about the sources available for treatment in their respective communities.

(*c*) Working for better hospital and clinical facilities.

(*d*) Promoting means of co-operation between Government, Provincial, Municipal and private Welfare agencies.

The local societies on alcoholism provide a valuable coordinating force between bodies in a particular community dealing with any aspect of alcoholism. They will not in any way compete with these existing agencies. Alcoholics Anonymous in particular will be regarded as a valued ally. A.A. will continue its rehabilitative work while the societies on alcoholism will develop services which A.A. is unable to render.

National Council office-bearers and executive: President—R. P. T. Anderson; *Vice-Presidents*—G. A. C. Kuschke and W. J. B. Slater; *Chairman*—Dr. R. A. Mathews; *Hon. Secretary-Treasurer*—Dr. G. M. du Plessis; *Executive members*— Drs. W. H. Alkema, M. C. Frame, B. Serebro, H. E. van Hoepen and C. B. Jeppe, Mrs. L. Swift and Messrs. E. J. Bevan, C. Bekker, A. Gilfillan and P. O'Foley.

The conference provided the opportunity for an interchange of ideas between the various societies. Members learned with interest of the virile Alcoholism Information Centre operating in Cape Town, which has handled over 2,000 interviews since its opening 15 months ago. Plans for a Foster Home and Information Centre in Durban were discussed. The East Rand society has secured a piece of land there and is planning to erect a small in-patient clinic.

Delegates took the opportunity of visiting an open meeting of Alcoholics Anonymous, 'Mount Collins' for women alcoholics and the group-therapy sessions at 'the Gables' (Toc H).

OFFICIAL ANNOUNCEMENT : AMPTELIKE AANKONDIGING

MEDICAL ASSOCIATION OF SOUTH AFRICA : MEDIESE VERENIGING VAN SUID-AFRIKA

FEDERAL COUNCIL

Notice is hereby given that a meeting of the Federal Council will be held at the Maccauvlei Country Club, Vereeniging, on 11, 12 and 13 April 1956, commencing at 9.30 a.m.

Agenda

1. Notice convening the meeting.
2. Proxies.
3. Minutes of previous meeting (circulated).
4. Matters arising out of the minutes.
5. Financial statement by Honorary Treasurer.
6. Report of the Executive Committee.
7. Reports of other Committees.
8. Reports deferred from previous meeting.
9. Notices of motion transferred from previous meeting.
10. New notices of motion.
11. Other business.

A. H. Tonkin
Secretary

Medical House
Cape Town
7 February 1956

FEDERALE RAAD

Kennis geskied hiermee dat 'n vergadering van die Federale Raad gehou sal word by die Maccauvlei Country Club, Vereeniging, op 11, 12 en 13 April 1956, aanvang 9.30 vm.

Agenda

1. Kennisgewing wat die vergadering belê.
2. Volmagte.
3. Notule van die vorige vergadering (reeds uitgestuur).
4. Sake wat uit die notule voortspruit.
5. Finansiële verslag van die Ere-Penningmeester.
6. Verslag van die Uitvoerende Komitee.
7. Verslae van ander Komitees.
8. Verslae van vorige vergadering oorgehou.
9. Voorstelle waarvan kennis op vorige vergadering gegee was.
10. Nuwe kennisgewings van voorstelle.
11. Ander sake.

A. H. Tonkin
Sekretaris

Mediese Huis
Kaapstad
7 Februarie 1956

PASSING EVENTS : IN DIE VERBYGAAN

The Third Acqui Prize in Rheumatology. This competition will be concluded at a meeting to be held at Acqui, Italy, in June 1957. Members of the medical profession specially interested in rheumatology as practitioners, lecturers or students and who desire to enter the competition or to receive a copy of the prize thesis should communicate with the Italian Embassy, 2 Grey's Pass, Gardens, Cape Town, not later than the end of March 1956.

* * *

The 'Jooste Cup' Golf Competition, organized by the East Rand Branch of the Medical Association of South Africa, and open to all members of the Association will be held this year at the Benoni Country Club on 15 April. This event is under the patronage of the Mayor of Benoni Councillor M. Nestadt, who will present the prizes. The competition is being held in Benoni in honour of their Jubilee Celebrations, and each competitor will be presented with a medal commemorating the occasion. Entires should be submitted to the convener, Dr. W. Sacks, P.O. Box 813 Springs (telephone: rooms 56-5371, residence 56-1138).

* * *

Dr. Zalmon Wolf, M.B., B.Ch., D.P.M., is now practising as a Neurologist and Psychiatrist at 53 Pasteur Chambers, 191 Jeppe Street, Johannesburg. Telephone: rooms 23-7679, residence 41-1469, emergency 22-4191.

Dr. Zalmon Wolf M.B., B.Ch., D.P.M. praktiseer nou as Neuroloog en Psigiater te Pasteurgebou 53, Jeppestraat 191, Johannesburg. Telefoon: spreekkamers 23-7679, woning: 41-1469, dringende oproepe: 22-4191.

* * *

Symposium on Venereal Diseases and Treponematoses. This symposium, sponsored by the US Public Health Service and WHO is to be held in Washington, D.C. from 28 May to 1 June 1956 (immediately following the 9th World Health Assembly at Geneva in May). The Surgeon General of the said Service invites V.D. control officers from national and other health agencies and various specialists from universities, research institutions, professional associations, etc. to attend. The purpose of the symposium is an international exchange of ideas and information on the latest developments in research, diagnosis, treatment and case-finding; it will include papers, panel discussion and general discussions.

The broad subjects to be covered include: (1) Control of V.D. and treponematoses, (2) Reporting and statistical problems, (3) Experimental syphilis and treponematoses, (4) Natural history of syphilis, (5) Serology and immunology, (6) Diagnosis, management and prognosis, (7) Gonorrhoea, minor V.D.s. and non-gonococcic urethritis, (8) Treatment, (9) Epidemiology and control techniques, (10) V.D. education, (11) The role of voluntary agencies, (12) cultural and socio-economic aspects.

Enquiries and notices of intention should be directed to Dr. L. A. Scheele, Surgeon General of the Public Health Service, U.S. Department of Health, Education and Welfare, Washington 25, D.C., USA (and a carbon copy to Dr. M. G. Candau, Director General, World Health Organization, (Geneva).

* * *

The Medical Graduates Association of the University of the Witwatersrand will hold a postgraduate refresher course in Obstetrics and Gynaecology on 15, 16 and 17 March. The course will consist of special demonstrations and lectures, ward rounds and symposia, to be held at the Medical School, the Queen Victoria Hospital and the General Hospital, Johannesburg. A detailed programme of the course is available on application to the office of the Medical Graduates Association, Medical School, Hospital Street, Johannesburg; telephone 44-7040 (mornings). As this course promises to be very popular early application is essential.

* * *

Research Forum, Groote Schuur Hospital, University of Cape Town. A meeting of the Research Forum will be held in the A Floor Lecture Theatre, Groote Schuur Hospital, Cape Town, on Tuesday 6 March at 12 midday, when Dr. R. Singer will talk about 'The Distribution of the Coronary Arteries in Bantu Hearts'.

The Research Forum will, in general, be held on the 1st Tuesday in every month during University term throughout 1956. General practitioners and other workers outside Groote Schuur Hospital are cordially invited.

* * *

International Medical And Surgical Congress. According to information supplied by the journal *Minerva Medica*, the Periodical International Medical and Surgical Congress will be held at Turin, Italy, on 1-9 June 1957. It is expected that the congress will include sections on Photobiology, Nuclear Energy, Automatism, Psychotechnics and Psychology of Labour, Neuroendocrine Surgery, Tumours, Infectious Diseases, Goitre, and other subjects. It will be associated with the Third International Medical and Scientific Film Festival and the Third Exhibition of Sanitary Arts. The Congress and Exhibition will be held at the Palace of Expositions at the Valentino, Turin, Italy. Further information may be obtained from the editorial offices of *Minerva Medica*, Casella Postale 491, Turin, Italy.

BOOK REVIEWS : BOEKRESENSIES

RURAL HOSPITALS

The Rural Hospital: Its Structure and Organization. By Dr. R. F. Bridgman. Pp. 162, with illustrations. Geneva: World Health Organization. 1955.

Contents: 1. Facts about Rural Areas. 2. Theory of the Rural Hospital. 3. Structure of the Rural Hospital Health Centre. 4. Architecture and Equipment of the Standard Rural Hospital Health Centre. 5. Staff. 6. From Theory to Practice. Bibliography. Index.

Dr. Bridgman, who is Deputy Director of Health of the Department of the Seine, France, has undertaken this study on behalf of the World Health Organization. He has collected a great deal of information, much of it interesting and some of it useful. He accepts the principle accepted by competent authorities everywhere that in rural areas the hospital should serve both a preventive and a curative purpose.

This was accepted by a conference of Far Eastern Countries in 1937 and had previously been accepted by the first Pan-African Health Conference, held at Cape Town in 1932.

The rural hospital advocated by Dr. Bridgman is a combined hospital and health centre, an ideal institution for South African conditions, but not possible while Central Government, Local Authorities and Provincial Administrations are responsible for different aspects of the same health problem.

The chapter on Architecture and Equipment is useful and some of the plans, made available by the US Public Health Service, can be made use of in this country.

This little book will be of interest to many. It should be read by hospital planners and administrators, and by sociologists and all interested in sociology. It is particularly recommended to missionary doctors, who may have to advise on the building, equipment and management of missionary hospitals.

R.S.

CLINICAL BACTERIOLOGY

Clinical Bacteriology. By E. Joan Stokes, M.B., B.S., M.R.C.P., M.R.C.S. Pp. 288 + vii, with 25 illustrations. 20s. London: Edward Arnold (Publishers) Ltd. 1955.

Contents: 1. The Practice of Clinical Bacteriology. 2. General Procedure. Records. Reports. 3. Culture of Specimens Normally Sterile. 4. Specimens from Sites with a Normal Flora. 5. Identification of Bacteria. 6. Investigation of Tuberculosis and Fungous Infections. 7. Antibacterial Drugs. 8. Clinical Immunology. 9. Hospital Epidemiology. 10. Media-Testing and Other Techniques. Index.

From time to time the advent of a less orthodox publication stirs the reader's interest. However, in order to merit a place on one's shelf, a technical book cannot rely on the trick of unorthodoxy; and Joan Stokes's codification of clinical bacteriological procedures avoids this trap.

It must be accepted that this publication is primarily for the practising bacteriologist and his senior technical assistants, it nevertheless contains rewarding information for all who are interested in bacteriology.

The academic bacteriologist is orientated to avoid false reasoning in reporting. An example cited suggests that it is more informative to report on a specimen of faeces to a clinician that 'no organisms of the Salmonella or Dysentery groups were isolated' than to report that 'cultures yield *Bact. coli, Strept faecalis* and *Proteus vulgaris*. The author states: 'Naming the species is at first sight impressive, but a full identification of them cannot be made without delay and it misleads the clinician to believe that those are the only viable bacteria in the specimen'. The sophistry of reporting on a specimen of faeces, 'no pathogens isolated', is well emphasized.

In South Africa, where the socio-economic spectrum of the bulk of the population is different from that in England, for instance, it is necessary that routine bacteriology should be disciplined to economic and adequate procedures, unnecessary elegance has little place in routine public health bacteriology. It is for this reason that it is pleasing to note this reference to correct reporting, and also to the legitimate employment of clinical findings.

With regard to the latter point, to secure the economy which stems from the correct laboratory procedure for the diagnosis and follow-up of pulmonary tuberculosis it is necessary to provide relevant clinical data with specimens of sputa sent in for examination. It is the reviewer's opinion that a laboratory may reasonably refuse to examine specimens that are not accompanied by clinical information.

In this book one readily comes across pieces of practical information which in text-books require close searching for. It is for instance a notorious fact that bacteriological reports on eye-swabs are commonly unsatisfactory. The author draws attention to the fact that bedside platings of eye-cultures are required in order to overcome the influence of lachrymal lysozyme.

The references to media testing, the inhibition of overgrowth of non-streptococcal organisms, or the preparation of a constant-dropping pipette, are surface plums for the picking.

There are, of course, omissions in the contents of the book and there are doubtful recommendations. For instance, in South Africa at any rate, it would be unrealistic to accept the recommendation that, 'in laboratories where ten or more (sputum) examinations are made daily there is no doubt that the fluorescent method is to be preferred'. A more significant criticism which may be levelled is that neither the section on clinical immunology nor that on hospital epidemiology are of wide appeal—the latter for an obvious reason, and the first because of its omission of a screen precipitin test or a standard Kolmer test for the serological diagnosis of syphilis.

All told, this is certainly a book well worth buying for the laboratory.

L.A.

PATHOLOGY FOR THE SURGEON

Pathology for the Surgeon. By William Boyd, M.D. (Edin.), Dipl.Psychiat. (Edin.), F.R.C.S. (Canada), F.R.C.P. (Lond.), M.R.C.P. (Edin.), F.R.S. (Canada), LL.D. (Sask.), D.Sc. (Man.), M.D. (Oslo). Seventh Edition. Pp. 737 + vi, with illustrations. Philadelphia and London: W. B. Saunders Company. 1955.

Contents: 1. The Surgeon and the Pathologist. 2. Inflammation and Repair. 3. Wound Infections. 4. The Granulomata. 5. Gangrene. 6. Shock and Burns. 7. Coagulation. Thrombosis and Embolism. 8. General Pathology and Tumours. 9. The Mouth and Jaws. 10. Neck, Salivary Glands and Esophagus. 11. The Thyroid Gland. 12. The Stomach and Duodenum. 13. The Small Intestine. 14. The Colon and Rectum. 15. The Vermiform Appendix. 16. The Gall Bladder and Liver. 17. The Pancreas. 18. The Peritoneum. 19. The Upper Urinary Tract. 21. The Male Reproductive System. 22. The Female Reproductive System. 23. The Breast. 24. The Spleen. 25. The Lympathic System. 26. The Cranium and its Contents. 27. The Spine, Spinal Cord and Nerves. 28. The Bones. 29. The Joints. 30. The Soft Tissues. 31. The Skin. 32. The Endocrine Glands. 33. The Cardiovascular System. 34. The Lungs and Mediastinum. Index.

All who use and enjoy Boyd's *Surgical Pathology*, will use with even more enjoyment this book which is its direct successor. The new title calls attention to a different approach to the subject in the mind of the writer, but postgraduate students, registrars, and surgeons will recognize an old friend in new guise, and much improved thereby. It does, however, take time to become accustomed to the increasingly common fashion of a double-column page.

The illustrations have been improved in quality and increased in number. This applies particularly to the section on 'Spine, Spinal Cord, and Nerves', where the text is also more informative. The paragraph on Injury and repair of nerves is greatly improved. 'The Mouth and Jaws' chapter is greatly improved over the parallel 'Jaws' in the final edition of *Surgical Pathology*. 'Burns' has been modernized satisfactorily, although the paragraph on Radiation burns is at once too brief and too generalized to be truly informative.

The position and usefulness of the frozen sectionist is judiciously set forth in an excellent and sensitive discussion, which should be read, marked and inwardly digested by any surgeon clamouring for this aid to diagnosis. Boyd states in this connection that he 'knows of no clinical nor experimental evidence' which shows that incision into a cancer (for biopsy) increases the occurrence of metastases. *Vox auctoritatis vox Dei*—or just *Boydi?* A noteworthy statement, anyway.

There are new chapters on 'Skin' and 'Lungs and Mediastinum'. A useful chapter on the 'Endocrine Glands' replaces that on the 'Middle Ear' and thus removes this hybrid onion from the pathological petunia patch. Naturally, the paragraphs on the 'Adrenals' benefit most from this replacement.

Some minor lapses, Kienböck for Keinböck, 'case' for 'cast' on p. 644, and 'foreign bodies' for 'loose bodies' on p. 585, will doubtless be corrected in future editions, for which there will certainly be a continuing demand.

R.D.H.B.

PATHOLOGY YEAR BOOK

The Year Book of Pathology and Clinical Pathology (1954-1955 *Year Book Series*). Edited by William B. Wartman, B.S., M.D. Pp. 486, with 168 illustrations. $6.00. Chicago: Year Book Publishers, Inc. 1955.

Contents: 1. Introduction. 2. General Pathology. 3. Cardiovascular System. 4. Hemopoietic System. 5. Respiratory System. 6. Alimentary System. 7. The Liver. 8. The Pancreas. 9. Urinary System. 10. Genital System and Breast. 11. Endocrine Glands. 12. Musculoskeletal System. 13. The Nervous System. 14. Hematology. 15. Clinical Microbiology. 16. Clinical Chemistry. 17. Electrophoresis, Chromatography and Proteins. 18. Ammonia Metabolism. 19. Enzymes. Miscellaneous Topics.

The latest edition of this series embraces the period 1954-1955. The book covers the recent advances in the whole field of Pathology, and the Editor, William B. Wartman, D.S., M.D., has devoted the first 69 pages to General Pathology and then divided his material under the various systems of the body. The final part of the book, some 150 pages, covers advances in Clinical Pathology.

In the opening part of the book, apart from a printer's error on p. 25 (reduplication of a sentence), the subjects summarized read well. An impression obtained, however, is that some of the articles could have been dealt with a little more fully; but for those further interested the references to all the original articles are given; as well as a fairly comprehensive index.

An interesting point to the reviewer was an account of 2 cases of fatal generalized B.C.G. infection in man, following on vaccination, reported from the University of Bergen. In the editors words: 'This case and the one previously reported show that B.C.G. is not always innocuous. In addition there are a certain number of cases of local abscesses, some of spread to regional lymph nodes and a few of skin lesions'.

The book is well printed and the photographs and photomicrographs are of good quality. It serves its purpose well, which is essentially that of a summary of recent advances in the last two years in Pathology. A minor criticism is the brevity of some of the summaries, and further that one or two of the articles could rather be classed as 'interesting' rather than 'advanced'. All in all, this book should provide points for those unable to devote the time to reading all the recent literature on this subject.

P.E.W.

RECENT MEDICAL AND HEALTH LEGISLATION

Recent Medical and Health Legislation. Supplement to Medical and Health Legislation in the Union of South Africa. By E. H. Cluver, *K.St.J., E.D.*, M.A., M.D. (Oxon.), D.P.H. (Eng.), F.R.S.I. Pp. 348 + xii. 27s. 6d. South Africa: Central News Agency Ltd. 1955.

Contents: 1. Medical, Dental and Pharmacy Amendment Acts. 2. The Nursing Act. 3. The Food, Drugs and Disinfectants Act. 4. Public Health Amendment Act 44 of 1952. 5. International Sanitary Regulations Act. 6. The Post Mortem Examinations and Removal of Human Tissues Act. 7. The Dental Mechanicians Act.

In 1949 Dr. Cluver published a much needed book covering all the medical and health legislation of the Union of South Africa. This valuable publication needed supplementation because of the many amendments, additions, and entirely new enactments. The new publication of Dr. Cluver's is a comprehensive supplement to his previous publication.

Each section is prefaced by a short commentary which covers and explains many of the important changes and additions. The contents of the book are indexed in great detail.

The new legislation covers a wide field, including laws relating to the training of medical practitioners, dentists, and chemists and druggists, and the practice of medicine, dentistry and pharmacy. It includes important sections on internships and the registration of medical practitioners visiting the Union. The control over harmful and habit-forming drugs has been tightened up. There are new laws concerning the Medical Council, professional fees, and the suspension, or restriction from practice, of registered persons.

The book covers amendments to the Nursing Act, extensive changes and additions to the Foods, Drugs and Disinfectants Act and changes in the Public Health Act which refer to the constitution of the National Health Council and refunds to local authorities resulting from recommendations of the 'Havenga' Committee. The latter incidentally led to the pegging of salaries of health officials which is now causing so much discontent.

There is an important chapter on International Sanitary Regulations which bring the Union into line with international sanitary regulations adopted by the World Health Assembly.

The Dental Mechanician Act is also brought up to date.

It is hoped that Dr. Cluver will rewrite his original book and bring it up to date. This is much needed, particularly in regard to the regulations made under the Foods, Drugs and Disinfectants Act, which have frequently been amended and re-amended over the last few years and now really require to be written up as a new set of regulations. Their present arrangement is confusing.

All doctors and members of relevant 'allied' professions should be in possession of Dr. Cluver's original book and this new publication. For medical officers of health and health administrators they are essential.

H.N.

CANCER FOR THE LAYMAN

One in Six. An Outline of the Cancer Problem. By I. Hieger, D.Sc. Pp. 80, with illustrations. 12s. 6d. London: Allan Wingate. 1955.

Contents: 1. Cancer: its Mythology and Reality. 2. Some Achievements of Cancer Research: The Statistical and Experimental Approach. 3. Cancer and Ageing. 4. Lung Cancer: Smoking and Smoke. 5. Cancer Theories. Epilogue. Acknowledgements. Glossary.

This book, by a distinguished biochemist, provides a readable and attractively illustrated outline of the cancer problem for the enlightened layman. The statistics are up to date and reliable, although the discussion of hepatoma in the Bantu fails to emphasize the limitations of statistics derived from miners, who represent a very young population, far from characteristic of the total Bantu population.

The description of experiments with carcinogens bear the stamp of first-hand acquaintance with the subject. The chapter dealing with cigarette smoking and lung cancer is convincing, the author blandly suggesting that cigarette factories should be closed so that the subsequent curve of lung-cancer deaths may provide the final evidence.

The title has been based on the present death-rate from cancer in England and Wales; the conclusion in the epilogue that some forms of cancer could largely be avoided by hygienic measures needs wider recognition, for certain cancers provide as valid a claim to the attention of public-health authorities as the acute infectious fevers.

The book can be recommended to any, expert as well as non-expert, who wish to obtain a summary of modern research into cancer.

A.G.O.

POLYCYTHAEMIA

Modern Medical Monographs: Polycythemia. Physiology, Diagnosis and Treatment Based on 303 Cases. By John H. Lawrence, M.D., D.Sc., F.A.C.P. Pp. 136 + viii, with 38 illustrations. $5.50. London and New York: Grune & Stratton, Inc. 1955.

Contents: 1. Polycythemia Vera. 2. Relative Polycythemia. 3. Secondary Polycythemia. Summary. Case Histories. References. Index.

Monographs on a single disease, written by authorities who have spent many years studying it, have become more and more popular in recent years. Parkes-Weber wrote one on this topic in 1922 and the present booklet will help to bring the subject up to date. Dr. Lawrence is well qualified to write it since he has been able to base it on a study of 303 cases. The pathology and clinical picture of the various types of polycythaemia are well described. The section on treatment is very good and what one would expect in view of the author's extensive experience with the use of 32p. The description of the author's original work on the polycythaemia of high altitude is also noteworthy.

There is much to criticize. It is not an 'almost forgotten fact' that polycythaemia vera is nearly always characterized by hyperplasia of the myeloid as well as the erythroid elements of the bone marrow. The fact is mentioned in most text-books on haematology. The relationship of polycythaemia vera to myeloid leukaemia, myelosclerosis and myelofibrosis is not well handled. There is no definition of the polycythaemic level (and no clear distinction between males and females); nor is 'leukaemia' defined and there is no mention of the concept of non-leukaemic myelosis or the erythroleukaemic chain. A few immature precursors of red and white cells in the peripheral blood do not constitute leukaemia. It is difficult to accept that 20% of patients with polycythaemia vera have or develop an associated leukaemia.

The whole question of the diagnosis of polycythaemia vera is a difficult one, more especially the differentiation between this disease and relative and secondary types of polycythaemia. One gets the impression that the authors have over-simplified the issue. Blood-volume studies appear to be essential, but even some of the cases 'of relative polycythaemia' appear to have a high true red-cell volume (Table 4). One hopes that the iron utilization will help, but so far this has not become universally applicable.

One does not expect a full review of the literature in a monograph of this type, but it surely should not omit mention of a critical review published in recent years in a South African journal.

Finally, measurements of red-cell survival in polycythaemia vera, published before the contributions in this field of the author's associates appear to have been omitted. This is a regrettable oversight.

C.M.

HAEMOPHILIA AND RELATED CONDITIONS

The Diagnosis and Treatment of Haemophilia and its Related Conditions. Medical Research Council Memorandum No. 32. By R. G. Macfarlane and Rosemary Biggs. Pp. 22. 2s. 6d. London: Her Majesty's Stationery Office. 1955.

Contents: 1. Introduction. 2. Clinical Diagnosis. 3. Laboratory Diagnosis. 4. Technique: (*a*) Preparation of Special Reagents; (*b*) Preliminary Tests; (*c*) Tests on Samples with an Abnormal One-Stage Prothrombin Time; (*d*) Tests on Samples with a Normal One-Stage Prothrombin Time. 5. Treatment. References.

In recent years rapid progress has been made in the study of the haemorrhagic disorders, and many clinicians and laboratory workers have felt bewildered and puzzled when faced with a patient presenting with hereditary or other haemorrhagic diatheses. The Medical Research Council of Great Britain have realized this position and they have asked Dr. R. G. Macfarlane and Dr. Rosemary Biggs to prepare this short memorandum. They could have made no better choice.

This 22-page booklet maintains the high standard we have come to expect from the Medical Research Council's publications. It covers the clinical and laboratory diagnosis of these haemorrhagic states. There is a fairly detailed technical section which is quite adequate as a guide to most laboratory workers, and also a short section on treatment. Most of the work has been published previously but it has never before been made so conveniently and inexpensively available.

The advice which is given is concise, accurate and authoritative. It is simply set out, easy to follow and easy to understand. The memorandum will receive a warm welcome from clinicians, pathologists and medical students.

C.M.

MAYO CLINIC PAPERS

Collected Papers of The Mayo Clinic and the Mayo Foundation. Edited by Richard M. Hewitt, B.A., M.A., M.D. *et al.* Volume XLVI. Pp. 843 + viii, with illustrations. Philadelphia and London: W. B. Saunders Company. 1955.

Contents: 1. Alimentary Tract. 2. Genitourinary Diseases. 3. Ductless Glands. 4. Blood and Circulatory Organs. 5. Dermatology. 6. Head, Trunk and Extremities. 7. Thorax. 8. Brain, Spinal Cord and Nerves. 9. Radiology. 10. Physical Medicine and Rehabilitation. 11. Anaesthesia, Gas and Intravenous Therapy. Miscellaneous. Indices.

During the past year no less than 629 papers were published by members of the staff of the Mayo Clinic. In this volume 134 of them have been reproduced, some in full, others in abstract.

The articles are presented in sections according to the anatomical system concerned. The first chapter contains papers dealing with the Alimentary Tract and here one finds an excellent review of the therapy of ulcerative colitis by J. A. Bargen. It is interesting to note the author's complete lack of enthusiasm for steroid therapy in this condition. Other commendable papers deal with hiatus hernia, functioning ovarian tumours and the management of thyrotoxicosis, myocardial infarction and renal disease.

At the Mayo Clinic thyroidectomy is performed under local anaesthetic. By this means, the risk of permanent damage to the recurrent laryngeal nerves is thought to be reduced. The results obtained would seem to justify the procedure.

The whole volume is essentially practical and clinical, and does not include any highly technical or abstruse articles. It may be regarded as 'light' medical reading, which should be enjoyed by all members of the profession.

R.H.

CORRESPONDENCE : BRIEWERUBRIEK

RELAXANTS IN ANAESTHESIA

To the Editor. I noted with interest the editorial[1] in the *Journal* on 'Safety First in Anaesthetics', and the most effective criticisms of it by Dr. Roberts[2] and Dr. Kok.[3] The experience of this department, which pioneered the introduction of d-tubocurarine chloride into English anaesthetic practice in 1946, has been that the greatest safety in anaesthesia is obtained by the correct use of muscle relaxants. Clinical results obtaining here are in direct contradiction to those of the American authors (Drs. Beecher and Todd[4]) upon whose work your editorial was based.

It is a great pity that your editorial did not consider the critique of Beecher and Todd's work that appeared in a later issue of the same journal, signed by 16 prominent American anaesthesiologists[5] from the New England states in which Prof. Beecher's department is situated (Harvard Medical School, Boston, Mass.). These authors found fault with the method of statistical analysis, and the conclusions drawn therefrom; in particular, they consider the 'curare' death rate presented to be 'completely misleading'. In fact, they wrote: 'The undersigned anesthesiologists find it necessary to present publicly an adverse criticism of this article, in order to counteract what we believe may be a disservice to anesthesiology and to the patient'.

If any conclusion can be drawn from this statistical jungle, it must surely be a criticism of American practice, and not of drugs which appear to be used with safety elsewhere.

From his writing, Prof. Beecher is an exponent of a belief of an inherent 'toxicity' of muscle relaxants. Perhaps it is unfair to suggest deliberate bias in such an article, even though this 'toxicity' is mentioned. There is no reliable clinical or experimental evidence to support this idea; the evidence available indicates that d-tubocurarine chloride has no effect on vasomotor control, no effect on the myocardium, no effect on cardiac output, does not pass the blood brain barrier in therapeutic doses, and that its effect can be safely reversed with atropine followed by neostigmine. The heart may also be protected from arrhythmias of vagal origin. These facts may not all apply to the miscellany loosely termed 'curare' by the American authors, which is a pity since their article might then at least have had the merit of indicating which relaxant affords the greatest safety in use.

From my experience of anaesthetic practice in Boston it is possible to gain some insight into the reason why relaxants have achieved an undeservedly bad reputation. There was a reluctance to protect the patient's airway by endotracheal intubation, adequate assistance of inadequate respiration was often not practised, and unnecessarily deep planes of anaesthesia were often maintained with ether or cyclopropane. This last habit introduces the hazard of depressing the vasomotor control that is essential in ensuring

adequate venouss return to the heart during assisted or controlled respiration, especially following blood loss. (Here in Liverpool it is found unnecessary to use anything more than nitrous oxide with relaxants during the maintenance of anaesthesia for intra-abdominal and intrathoracic surgery.) Lastly, there seemed to be a tendency to avoid the use of neostigmine to reverse relaxation at the end of operation, thus adding greatly to post-operative dangers.

It was suggested to me by a thoracic surgeon who recently visited some of the hospitals whose figures are quoted in Beecher and Todd's report that the standard of anaesthesia was frequently impaired because the surgeon dictated the anaesthetic technique to be used, when his anaesthetist was usually far better qualified to select the best method.

P. A. Foster

Assistant Lecturer
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48 Bedford St. North
Liverpool
18 February 1956.

1. Editorial (1955): S. Afr. Med. J., **29**, 1084.
2. Roberts, F. W. (1955): *Ibid.*, **29**, 1211.
3. Kok, O.V.S. (1956): *Ibid.*, **30**, 95.
4. Beecher, H. K. and Todd, D. P. (1954): Ann. Surg., **140**, 2.
5. Abajian, J. *et al.* (1955): *Ibid.*, **142**, 138.

RELAXANTS IN ANAESTHESIA

To the Editor. Does Dr. Samson[1] really believe that it is common knowledge throughout the medical profession that the mortality of anaesthesia in South Africa has increased 6-fold with the use of the muscle relaxants? I do not wish to be accused again of misquoting, but that seems to be the inference from the first two sentences of Dr. Samson's letter in your issue of 11 February 1956.

Dr. Samson does not agree with my complaint that your editorial[2] of 19 November 1955 was unfair, although he quotes accurately my reason. It is not clear why he disagrees, because I also say the figures from Beecher and Todd are very alarming. The unfairness lies in not putting the other and generally-held side of the picture.

Dr. Samson next accuses me of quoting you 'out of context and somewhat inaccurately'. I do not know what he means by 'out of context'. As for 'somewhat inaccurately', he fails to support his serious allegation with any example of inaccurate quotation from your editorial. I gather that he objects to the use (when quoting Beecher and Todd, not your editorial) of plain print instead of the original italics for the word 'know'. If this is inaccuracy, I apologise, but the use of the italics does not alter the sense at all; it only emphasizes my point. If Beecher and Todd had admitted that they knew (with or without italics) that 'curare', by which they mean any relaxant, including Scoline, had caused the death of a patient, they would not have laid themselves open to a charge of culpable homicide. They were reporting the collected evidence from 10 general hospitals, not their own experiences, and in any case the *bona fide* use of a drug intended for the benefit of the patient, even if it proved to have the adverse effect, would not, I am sure, in the United States constitute grounds for a charge of culpable homicide. Even in this country, where a charge of culpable homicide is not unheard of against anaesthetists, I believe that it is necessary to prove negligence to substantiate such a charge.

Dr. Samson further quotes me, again quite accurately, that 'in practice no patient should die from hypoxia due to the use of relaxants or respiratory drugs.' I repeat this, and agree with Dr. Samson that unfortunately they do die. The point of my sentence was the word 'should'. Perhaps I ought to have put it in italics. Let me stress the point again that the fact that patients do die is the fault of the way the drugs are misused, or the resultant respiratory depression mismanaged, and not the fault of the drugs *per se*. That was the main point of my previous letter, and where I, happily in the company of the majority of informed pharmacologists and clinicians, am opposed to the views of Beecher and Todd.

Dr. Samson is 'of strong opinion that sudden cardiac arrest can be produced by a massive dose of a long-acting relaxant.' I hope I have quoted him accurately. I am not interested in the strong opinions; facts are what interest me, and theories to become facts must be substantiated by pharmacological and clinical proof. In any case we are not concerned with what could happen with problematical massive doses, but with what does happen with normal clinical doses.

In his last sentence, in spite of what goes before, the true object of Dr. Samson's letter is revealed: He is determined to be in vehement agreement with me. I will go one stage further than he does and suggest that both long-acting and short-acting relaxants should be abandoned by the *inexperienced*.

F. W. Roberts

309 Harley Chambers
Jeppe St.
Johannesburg
21 February 1956.

1. Samson, H. H. (1956): S. Afr. Med. J., **30**, 155.
2. Editorial (1955): *Ibid.*, **29**, 1084.

RELAXANTS IN ANAESTHESIA

To the Editor. Were Dr. Samson to omit the specific from his 'opinion that sudden cardiac arrest can be produced by a massive dose of a long-acting relaxant', the generalization might be a suitable introduction to a students' Pharmacopoeia. It would certainly apply forcibly to the entire range of anaesthetic agents and would justify, rather than detract from, modern methods of 'balanced anaesthesia'. To invest the relaxants, which *par excellence* produce required effects with a minimal, of not complete, absence of side-effects, with a mumbo-jumbo of unproven 'toxicity' is indefensible.

In the unconscious state, maintenance of a satisfactory airway—implying avoidance of hypoxia and hypercarbia — is a recognized prerequisite of survival. The relaxants are a safe and potent aid to obtaining such airway, not merely in intubation, but in the prophylaxis of vagomimetic spasms of jaw, larynx and bronchi, of induction vomiting, and of the occasional all-in wrestling bout of pre-relaxant days.

Administration of a relaxant is usually coupled with that of an intravenous barbiturate, and it is a fact that even moderate doses of the latter may, in hypoxia, cause irreversible myocardial damage; as the same danger exists to some extent with all true *anaesthetic* agents, implication of relaxant aids in anaesthetic fatalities must fall in the 'misuse' category rather than 'dangerous'.

I feel it would be more reasonable to suggest that an anaesthetic administered in such a way as to produce death from anoxia would be rendered safer by reasonable use of relaxants to obtain controllability and a reduced metabolic rate.

The use of relaxants is associated with a technique based on primary physiological principles, and confidence in its practice is soon acquired. Surely it behoves all who administer more than the occasional emergency anaesthetic to familiarize themselves with methods proved vastly safer than the practices to which Dr. Samson would apparently have the G.P.-anaesthetist adhere?

R. G. Drummond

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S. Rhodesia
18 February 1956

KEROSENE (PARAFFIN) POISONING

To the Editor. I feel I should record my experience in treating sixty (60) cases of accidental ingestion of paraffin in young children aged 10 months to 2¼ years. Over the past 10 years I have employed gastric lavage (using a dilute solution of sod. bicarb. in every instance, the patient being held on an adult's lap in a sitting position with the trunk slightly flexed. On departure the parent is asked to offer only milk feeds for the rest of the day.

No medication of any kind has been prescribed, and there has not been a single case of residual respiratory illness clinically. The amounts of poison ingested varied from a 'tongue-touch' to ± 3 ounces. In August 1952 I gave the same treatment to an adult European who had the misfortune of ingesting half a bottle of the stuff (he had been a beer drinker), before he registered the taste. His Native servant had mistakenly placed the paraffin bottle instead of the water bottle in the refrigerator, and the patient had been doing some heavy gardening.

J. Salkinder

268 Pretoria Avenue/Laan 268
Ferndale
16 February 1956.